A TEXT-BOOK

OF

DERMATOLOGY

BY

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PREFACE TO THE SECOND EDITION.

The first edition of this book was exhausted several years ago. The Great War for some time absorbed all our activities and all our thoughts. But the time has come to think of the young physicians returning from the armies and make ready the means for facilitating the work they are called upon to perform. I have accordingly prepared this second edition, in spite of the difficulties of these troublous times. I have insisted upon its being entirely abreast of the progress of science and as complete as possible without unnecessary details. The general plan of the book, which has been tested and found to possess real didactic advantages has, of course, been retained.

Many paragraphs have required rewriting and more or less considerable elaboration. Among these may be mentioned those dealing with anaphylaxis, phagedena, the sarcoids, the gangrenes, the cutaneous atrophies, inguinal epidermophytosis and the dermatomycoses. The review of the general interpretation of tuberculosis, congenital syphilis and the xanthomata has had to undergo notable modifications; paragraphs or new chapters have been devoted to the dyskeratoses, cutaneous diphtheria, the leishmanioses, the cutaneous leukemias, the tophi of gout, etc. The introduction of the serodiagnosis of syphilis and the treatment with the arsenobenzols into daily practice has demanded the allotment of considerable space to these subjects. Finally, in rewriting the therapeutic notes, I have endeavored to make them still more practical and to emphasize the recommendations dictated by my personal experience as to the choice of medication and the errors to be avoided.

Professor Jadassohn, of Bern, has added to the German translation of this book which appeared under his direction in 1913, a considerable number of notes which I have carefully retained when-

ever his suggestions seemed to me to be of value for the reader; they have been incorporated in my text, of course, mentioning their origin.

As it stands, this volume although rejuvenated and somewhat better nourished, remains essentially the same as its predecessor. I can only wish that it may meet with the same kind reception. My excellent and devoted publishers have left nothing undone to present it in good shape and I herewith express to them my sincere and grateful acknowledgments.

J. Darier.

EDITOR'S PREFACE.

In undertaking the task of editing Darier's Précis de Dermatologie I have been actuated less by a personal friendship with the distinguished author extending over a period of thirty years, than by a sense of the importance of introducing to the English-reading student of dermatology a work that is in many respects unique in its presentation of the subject and, as it seems to me, of extraordinary value for the student and the teacher.

The plan of the work is fully explained in the author's introductory chapter and needs no further comment. The German translation of the first edition enjoyed the advantage of editorial comment at the hands of one of the foremost dermatologists of our time, Professor Jadassohn of Berne, and Darier has made use of these comments in this the second edition of his Précis. In preparing the work for English readers I have added notes which are enclosed in brackets, [ ] to differentiate them from the author's text and which, in the case of more extensive notes, are printed in a different style of type. It need not be emphasized that they are not intended by way of criticism: a few of them seemed to me desirable for the elucidation of the text; some were suggested by conditions peculiar to English and American practice and some convey my personal experience in clinical dermatology. It can hardly be expected that two dermatologists practicing their profession for thirty years in different hemispheres will be of the same mind on all subjects.

There is no dearth of excellent treatises of dermatology in the English language and this work may confidently be expected to find its place beside them. The student cannot fail to profit from a study of Darier's unusual power of delineation in brief compass, his presentation of clear-cut cameos of description, his obvious habit of thinking of lesions in relation to their anatomical structure and always with a background of general pathology. To these sufficient

reasons for producing another book may be added the author's originality in the arrangement of his subject-matter and finally the desirability of placing before English-reading students the views of a foreign author, especially one who represents an old and honored school of dermatology.

French dermatologists have played a leading role in the development of our science. The Hôpital Saint-Louis, the greatest dermatological clinic in the world, has afforded an extraordinary opportunity for study and for more than a century it has furnished a succession of great dermatologists who have handed down their experience from teacher to pupil in an unbroken chain. Berne, Berlin, Breslau, Hamburg, London and many cities in America and elsewhere have excellent dermatologists whose observations and studies have greatly enriched our science; but in all these centers, dermatology is the work of one man or of a number of independent units. Vienna indeed furnishes an example of a school of dermatology that has exercised a powerful and lasting influence in its field. But Vienna was a one-man school and unfortunately the great light shed by Hebra so blinded his followers that for a generation the Vienna school suffered from a form of dermatological amblyopia: neither Duhring's disease, for instance, nor pityriasis rosea were recognized in Vienna as dermatological entities. French dermatology is unique in the peculiar circumstance of a familiarity with the great masters of the past that is almost personal. Alibert, Cazenave, Biett, Dévèrgie, Bazin, Vidal, Fournier, Besnier, seem living personalities to the present-day student at the Saint-Louis. The advantages derived from this local continuity of observation and practice are inestimable.

There is, however, an inherent danger bound up with its advantages; that is the danger of falling to too great an extent under the influence of tradition. Forty years ago French dermatology was suffering from the defects of its merits and it is only recently that such conceptions as are implied in the terms herpetism, arthritism, lymphatism, etc., have ceased to sway French dermatological thought and traces of these conceptions still linger in French medical literature, side by side with the most advanced thought in medical science.

Another possible effect of tradition may be found in the current

French dermatotherapy. French physicians to a great extent are in the habit of employing elaborate and complicated formulas in their treatment of diseases. Most of these have the sanction of long usage; some of them are even medieval in their construction. French dermatologists probably have not less success in curing their patients than those of other countries and it is surely not less interesting than instructive to learn that there are various ways of accomplishing this result.

The observant reader will be struck with the great preponderance of references to achievements of French dermatologists. The assumption that this is an illustration of a narrow spirit which is out of place in a scientific work will be dispelled if the reader will examine an English, American, German or Italian text-book from the same point of view. He will find that this form of Chauvinism is common to all nations and is indeed perfectly natural. An author obviously will cite the literature with which he is most familiar and which is most accessible to his readers. From this point of view again it is an advantage to the English reader to become acquainted with the work of a foreign author and thereby acquire a broader outlook on his specialty.

The Editor ventures to express the hope that this work of Darier's will be found not less useful to the reader than it has been instructive and inspiring to him.

S. Pollitzer.

New York, 1920.

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DISEASES OF THE SKIN.

INTRODUCTION.

This book is destined for new students entering a dermatological service and for general practitioners whose hospital recollections have become somewhat indistinct. My object has, therefore, been to make it as concise and practical as possible, while including the entire domain of cutaneous pathology. In order to make it short, I have been obliged to sacrifice the entire bibliography, historical references, quotations, learned discussions, etc., limiting myself to the essential data from the standpoint of diagnosis and treatment. In order to make it practical, it has seemed to me desirable to arrange the material according to a plan which is not customary and which I must first explain and justify.

MORPHOLOGY OF THE DERMATOSES.

In the first part (Chapters I to XXII) the eruptive lesions and the non-eruptive cutaneous changes, namely, the *elementary dermatological forms* are discussed and to each of these a description of the principal *syndromes* in which the elementary forms occur is added. This requires some explanation. In dermatological practice, the physician is confronted by very peculiar conditions. He does not have to look for pathological symptoms with the help of more or less complicated devices; the symptoms present themselves directly; he recognizes their features and their localization at once and witnesses this development. Under these conditions it seems obvious to me that a book intended to facilitate the study of skin diseases should accord the first place to morphology, namely, *to what can be seen*. In regard to the visible manifestations of cutaneous pathology, two contingencies are possible: the condition may be an eruptive dermatosis, or the dermatosis under consideration may be non-eruptive.

A. Eruptive Dermatoses.—This term is applied to skin diseases made up of efflorescences, namely, spots, papules, vesicles, etc. The analysis of every eruptive dermatosis must take up succes-

sively four orders of facts: those relating to the efflorescence, to the eruption, to the disease and to the patient.

1. The *eruptive lesion* deserves attention in the first place. This is in reality merely the anatomical lesion of the dermatosis as it appears to the naked eye. To inspect attentively an efflorescence, to palpate it, to scratch it, to compress it under a glass slide, to prick it with a needle so as accurately to determine its type—this constitutes in a general way the naked-eye study of the pathological anatomy. Conversely, it may be stated that to subject the efflorescence to biopsy for the purpose of studying its histology means its clinical examination under the microscope.

The efflorescences are of fundamental importance; they really represent the dermatologist's alphabet and no one ignorant of them can learn to read the skin.

2. The *eruption* represents the efflorescences taken as a whole. It is visible and all its features are in evidence; it is necessary to note its abundance, its dissemination or its confluence, its topographical distribution, etc.

An eruption is said to be simple or pure when it is formed of lesions of the same kind and of the same degree of development; it is described as deformed, when the lesions are in different stages of evolution; it is called complex or polymorphous when it is made up of lesions of various types; complicated, when the primary lesions become associated with other lesions secondary and of another kind.

3. Information concerning the *disease* is usually furnished by inquiry; it is necessary to ask about the mode of onset, the preceding or accompanying circumstances, the extracutaneous symptoms, the course, etc. Direct examination, however, will often permit a series of data to be secured in this connection which will forestall the patient's answers and serve to control their accuracy.

4. Among the conditions peculiar to the *patient*, some are of an objective kind; for example, the sex, the age, the race and the constitution of the individual. Information must also be obtained in regard to his occupation, his hereditary, hygienic and pathological antecedents, etc.; the viscera, body fluids and functions must be reviewed; briefly, a general clinical examination be carried out in order to ascertain the nature of the soil on which the skin disease develops.

B. Non-eruptive Dermatoses.—In a second group of cases the physician is not confronted with an eruption. The cutaneous alterations may consist, for example, of a change in color of the skin, or dyschromia; in hypertrophy or atrophy; in a lesion of the nails, the hairs, etc.

This altered appearance of the integuments can sometimes be referred with a high degree of probability to a definite inflamma-

tory, degenerative or dystrophic *pathological process*. In other cases, however, it is impossible to determine the nature of the process. It therefore seems preferable to me to depend on what can be seen and to restrict oneself to classifying the pathological condition as it exists, instead of venturing into the ever-fluctuating domain of general pathology.

The apparently extremely variable morphology of skin diseases can thus be brought down to various forms of *eruptive lesions* on the one hand and of *pathological conditions* of the skin on the other. I have combined them all under the name of *elementary dermatological forms*. For the descriptition of dermatoses on the bases of their morphology, my work thus becomes reduced to the following:

Selection of a certain number of elementary forms, easily distinguished or recognized after very little study; description of these forms as accurately as possible, indicating the special anatomical lesions from which they result.

Next, in view of the fact that these dermatological forms may be encountered in various cutaneous affections, it still remains to take up these affections one by one and to show what constitutes their individuality and on what their diagnosis is based.

The dermatological types and cutaneous affections which can be approximated on the basis of their morphology are certainly not pathological varieties or diseases in the nosographic sense of the word, but are simple *syndromes*. Now these syndromes are of two different kinds. Some have an unknown or complex etiology (example: psoriasis); when they have been described with all their features, there is nothing more to be said. Others on the contrary are referable to a known specific cause (example: psoriatiform syphilides) and constitute one of the possible manifestations of a definite disease. The latter will have to be referred to again in the second part of this book and given their proper place under the general description of this disease.

The first twenty-two chapters are accordingly dedicated to the principal elementary dermatological forms and to the syndromes derived from them.

A classification of cutaneous diseases on the basis of their morphology presents this great advantage that the question of the diagnosis is presented under the same form in which it is met in dermatological practice. On the other hand, it is open to two principal objections. The first is this, that an attempt to take into account all the eruptive forms to which the same disease can give rise, would necessarily involve the splitting of its description into very many fragments. In order to avoid this difficulty, I have restricted myself in the first part to dwelling upon the most common and most characteristic syndromes; the others are briefly

mentioned so as to warn the reader against a possible mistake; their description will follow in the second part of this book.

The other objection is that an arrangement based simply on clinical appearances can in no way constitute a classification. It does not claim to do so. Nobody would think nowadays of renewing the attempts of Plenke and Willan. It is generally conceded that the only logical and scientific classification in dermatology as well as in other branches of pathology is that which is based on etiology.

NOSOGRAPHY OF THE DERMATOSES.

In the second part (Chapters XXIII to XXXI) the point of view is entirely different. There I review the diseases of the skin itself, the pathological entities with a definite etiology, classified according to the nature of their cause. Hence there will be found: *artificial dermatoses, parasitic dermatoses, infectious dermatoses,* etc.

I have put into this class a group of skin diseases united, if not by a first common cause, at least by an identical phenomenon of a general nature, namely, essential pruritus, dependent upon a nervous disturbance; this appears to be the second cause of the cutaneous manifestations. This group can be designated under the name of pruritus or *neurodermatoses.*

I finally group among the pathological entities the *tumors of the skin;* admitting that in doing so I conform with custom rather than follow a personal conviction. The etiology of the majority of tumors is unknown or theoretical; it is for this reason no less than on account of their morphology and course that they have hitherto been considered as forming a natural group.

The etiological classification of skin diseases is confronted with an insuperable difficulty. There exists a series of eruptions, by no means very rare or imperfectly characterized (for example, eczema, lichen, psoriasis) the etiology and pathogenesis of which are entirely unknown. The question arises what place should be assigned to these eruptions. The plan which I have adopted enables me to omit gratuitous hypotheses and avoid forced analogies. These eruptions, the causes of which are multiple, complex or unknown, are not diseases but pure syndromes. Their morphology is practically all that is known about them. They have been described in the first part and do not require to be mentioned in the second.

THERAPEUTIC NOTES.

Although I have carefully indicated the attitude to be maintained by the practitioner in connection with each dermatosis, I have deemed it advisable to add a brief therapeutic review to this book.

It contains the essential data required for dermatological treatment and the not very numerous prescriptions which it is indispensable to know. It frequently happens that these prescriptions are applicable to several classes of cutaneous affections; by uniting them at the end of the volume, I have avoided frequent repetitions.

Imperfect as it is, this little book is the fruit of long experience. I have practised and taught dermatology for more than thirty years. The plan was conceived and the text frequently revised in the hospital. By this statement I do not mean to claim that all its contents are entirely original. In the expression of an opinion or in the rendering of a description, it is impossible, at least for me, to separate what is personal from what I have learned from teachers, from the literature and from associates.

A book of this kind does not aim at replacing the classical treatises, but it is meant to serve as an introduction to and a summary of these larger works.

J. Darier.

PART I.

MORPHOLOGY OF THE DERMATOSES.

CHAPTER I.

ERYTHEMA AND THE ERYTHEMATA.

Erythema is a congestive redness of the skin, circumscribed or more or less diffuse, usually temporary, which disappears momentarily under pressure of the finger. Redness of the skin which does not comply with this definition, such as the following, is not designated as erythema:

Red spots resulting from the deposit of a coloring matter and disappearing on ablution. Peristent red spots of congenital origin, due to increase in size and number of the bloodvessels, but not to congestion; these are *vascular nevi* (p. 693).

Red spots which do not disappear under pressure of the finger; these are *cutaneous hemorrhages* (III).

Erythemas which are at the same time very extensive or generalized and very persistent, are commonly designated by the name of *erythroderma* (VI).

The term *macules* is reserved for erythematous and pigmentary but not cicatricial spots following on cutaneous lesions or eruptions of any kind (p. 322).

Erythema is the first and most common skin reaction produced by an external or internal irritant.

All acute eruptions and the great majority of chronic skin lesions are accompanied by reddening; this *associated* erythema is such an ordinary phenomenon that it only exceptionally requires to be taken into consideration.

Simple, more or less irregular erythema constitutes the eruptive feature of a large complex group of dermatoses known as the Erythemata. The majority of these affections are so imperfectly characterized, however, that it is usually not an easy matter to determine whether a given erythema is a symptom or a disease,

making it impossible to describe these conditions separately. The following, therefore, applies indiscriminately to erythema and to the Erythemata.

Varieties.—Depending on the apparent pathogenesis of the cutaneous congestion, it is customary to distinguish between active or arterial erythema, due to an increased blood supply, and passive or venous erythema, due to stasis.

Active erythema, resulting from a congestive or acute inflammatory hyperemia, is characterized by a bright pink color and a rise of local temperature. It is often accompanied by a sensation of heat or by itching. As a rule, it is ephemeral, lasting only a very few days.

Passive erythema, on the contrary, produced by stagnation of the blood in the small cutaneous veins and capillaries, is of a darker or purplish-red color, the local temperature is diminished; it sometimes gives rise to a sensation of stiffness, with or without itching, and is generally more or less persistent.

In a large number of cases, it is impossible to decide if an erythema is active or passive. On the other hand, it is much easier to distinguish varieties, on the basis of whether the erythema is deformed; or from the configuration and duration of the eruption.

An erythema is described as *simple* when the erythematous congestion is pure, that is, when the change of color of the skin is not complicated by any changes of its thickness, consistence and epidermic surface. Otherwise, the erythema is *deformed*. As a matter of fact, it frequently happens that the exaggerated hyperemia leads to secondary accessory lesions, which more or less modify the characters of the eruption; these must be interpreted as irregularities of the eruptive type.

Erythematous congestion may thus become associated with: intradermic edema (*urticarial erythema*); cellular infiltration, manifested by a hard superficial elevation (*papular erythema*) or by deep-seated nodules (*nodular erythema*); interstitial hemorrhage (*purpuric erythema*); early or delayed pigmentation (*pigmented erythema*); raising of the epidermis in vesicles or bullæ (*vesicular* and *bullous erythema*); finally furfuraceous or lamellar desquamation (*squamous* or *desquamative erythema*).

These irregularities may be sufficiently marked to constitute intermediary or true transition forms, connecting the erythema with urticaria (II), papules (VII), nodosities (XIV), purpura (III), the dyschromias (XVI), bullous dermatoses (X), and erythrodermas (VI). Sometimes, real diagnostic as well as nosographic difficulties arise.

When in doubt as to the classification of an eruption, either among the erythemata or among the dermatoses of another group, it is advisable to base one's judgment not exclusively on the objective

examination of a given eruptive feature, but on the eruption as a whole, its development and the nature of the underlying process. This rule, although excellent in certain cases, does not suffice in others; the following examples are illustrative.

In the affection described by Bazin under the name *erythema induratum of young girls*, histology has shown the tissue-induration to be due to a tuberculoid infiltration. The nature of the pathological process here proves the condition to be not an erythema, but a hypodermic nodosity belonging to the tuberculides. The erythematous bullous eruption known as *hydroa* sometimes appears as an irregularity or a variety of polymorphous erythema, or again seems to be closely related to certain forms of bullous dermatosis. It is therefore differently classified by different authors.

The **configuration and extent** of the erythema permit a distinction between the following types:

Scarlatiniform or Scarlatinoid Erythema.—This is characterized by a more or less general, bright and uniform redness. The red color may result from confluence of miliary or lenticular hyperemic spots, or it may be diffuse from the start. The eruption is ephemeral or more or less prolonged; it is often of medicinal origin (mercury, quinin, opium, etc.), or due to infection (rash preceding smallpox or occurring in the course of gonorrhea, diphtheria, puerperal [and other streptococcic] fevers, indefinite infections). There exists a relatively durable form, with general symptoms, *recurrent desquamative scarlatiniform erythema*, which connects this type of erythema with the primary erythrodermias.

Rubeoliform Erythema.—This is made up of small, occasionally confluent spots, with ragged or diffuse margins, rarely slightly elevated; desquamation is absent or insignificant. The eruption is discrete, regional or generalized and of variable duration. The term *roseola* is employed for nummular or lenticular spots. Four groups of roseola are differentiated:

(1) *Roseolar exanthematic fevers* (rubella, rubeola, seasonal roseola); (2) *Symptomatic infectious roseola* (syphilitic roseola, typhoid roseola or lenticular rose-spots, roseola of typhus, rash preceding smallpox, eruptions of cholera, cerebrospinal meningitis, certain septicemias); (3) *Medicinal roseolas* (Copaiva balsam, santol oil, turpentine, among the so-called balsam roseolas; the roseolas of quinin, antipyrin, odine, etc.); (4) *Emotional roseola*, which is not a true skin affection, but a physiological phenomenon. It consists of a transitory redness, arranged in spots or as a network, appearing over the chest, neck and shoulders of certain individuals on exposure of the person and identical with the emotional redness of the face.

Erythema in Patches and Figured Erythema.—These consist of congestive spots, patches or surfaces of irregular shape, discoidal or

of variable configuration (erythema *marginatum* or *annulare*, etc.). Although sometimes infectious, this type of erythema is more frequently of medicinal, serotherapeutic or autotoxic origin.

The **duration** of an erythema, especially the active variety, is very short; as a rule, from one to four days at most. Some last much longer, however, for example syphilitic roseola. In certain forms, there is a marked tendency to *recurrences*.

Congestive spots persisting for several months are sometimes designated as *erythema perstans*, but do not represent a definite pathological type. The presence of a manifestation of this kind should suggest conditions such as tertiary syphilitic erythema, lupus erythematodes, parapsoriasis, tuberculides, the spots of macular leprosy, premycotic erythema, and so forth. Under the name of *centrifugal annular erythema*, I have recently described an eruption which develops acutely, but nevertheless persists for many months on account of the constant renewal of fresh lesions. The primary urticarial spots become rapidly transformed into rings, prominent and solid to the touch like cords; their peripheral extension, which may amount to several millimeters daily and the fragmentation of the rings, give rise to arches or strands whose design remains lightly pigmented. This form of erythema is peculiar and very rare; in the reported cases, it was situated on the buttocks, the back, and the thighs.

Pathological Anatomy.—The clinical appearance of erythema, its configuration and the irregularities to which the eruptive element is subject, are explained to a certain extent by the pathological anatomy. The only essential lesion is the dilatation of the blood-vessels of the cutis, more particularly those of the papillary body. It disappears in the cadaver and is usually no longer recognizable in cross-sections of microscopical specimens.

The common rounded or oval configuration of the spots of active erythema is accounted for by the anatomical arrangement, this form being precisely that of the vascular territories of the skin supplied by the same afferent arteriole. Between these territories of direct supply, there exists an anastomotic plexus where the circulation of the blood is normally less active and where the blood has a tendency to accumulate in erythema due to stasis (*livedo annularis*).

Intense congestion may give rise to exudation of blood plasma from the vessels (urticarial erythema) and to diapedesis of white corpuscles mixed with a few red blood cells. These elements are deposited as cuffs around the vascular branches, the inflammatory increase of the fixed cells contributing to the production of induration and elevation (papular erythema).

The anatomical explanation of the pigmentation, phlyctenization

and desquamation, which are the possible results of erythema, will be found elsewhere.

Etiology.—The causes capable of producing erythema are extremely numerous and varied. An attempt has been made, rationally enough, to group erythema on the basis of its etiology, but this leads merely to an incomplete and artificial classification. the condition representing as it does, not a disease, but purely a symptom. One identical cause may give rise to erythema of a variable objective appearance and course; on the other hand, the same form of eruption may depend on several different causes acting separately or together. In a number of cases, a factor of primary importance, known as predisposition is involved.

The actual or immediate causes of erythema are either external or internal.

External Causes.—Erythema from direct provocation; any slight traumatism causing local hyperemia. The passage of the nail over the skin causes the transitory appearance of a red line; an exaggeration of this phenomenon acquires a positive diagnostic value in certain cases (*vasomotor or meningitic streak*). *Intertrigo-erythema* is in part of mechanical origin. The effects of repeated friction will be pointed out under the heading of the *artificial dermatitides* (XXIII). Caloric and medicinal external erythema is discussed in the same chapter. Erythema *à frigore* and active erythema will be referred to presently.

Bites or stings of the epizoa, lice, bugs, etc., of various insects, such as bees, wasps, hornets, mosquitoes and contact with the hairs of certain caterpillars or some plants, also give rise either to erythema or urticaria, of variable severity and extent in different individuals, or to a true inflammatory dermatitis.

Internal Causes.—Erythema may make its appearance following the ingestion of certain foods or medicinal agents: the *pathogenetic erythema of Bazin;* or in the course of various infections, specific or ordinary: *infectious erythema;* or finally, under the influence of individual conditions of a nervous, autotoxic, dyscratic or unknown kind, constituting a predisposition or idiosyncrasy.

Erythema of alimentary origin may be transitory and diffuse, occupying especially the face and the upper part of the trunk, or it may be more lasting. It assumes an urticarial or papular form, or that of a roseola or of marginate patches. All those food substances which are usually prohibited in urticaria and eczema, have been held responsible; although some of these really possess a certain degree of toxicity, the majority act undoubtedly only with the assistance of indigestion, habitual dyspepsia, or rather that of a predisposition which is now apt to be referred to *anaphylaxis* (pp. 460, 477).

Medicinal and *serotherapeutic erythema* belong under the heading of the toxidermas (XXIII).

Autotoxic erythema is the designation of conditions developing in uremia, gout, diabetes and hepatic diseases or in the course of acute or chronic appendicitis, obstinate constipation, etc. Such cases are referred to the production of autogenous poisons and insufficiency of the renal, hepatic, intestinal emunctories, etc. A popular belief to the effect that the skin under these conditions is damaged on account of the vicarious role it is made to play in substituting the organs intended for the normal purification of the organism, is shared by a number of physicians, on what grounds I do not know. This idea is purely theoretical and rests on no reliable basis.

Infectious erythema is observed in a large number of diseases, being due either to the specific microbe or to a secondary microbic association. As a rule, it is *toxic infectious*, the parasite acting apparently through the intermediation of its toxins or the resulting changes in the composition of the blood. Aside from the infections mentioned above, when speaking of roseola, mention must be made of the following, as capable of giving rise to erythema: Puerperal infection, ulcerative endocarditis, pneumonia, diphtheria, the anginas, vaccinia, gonorrhea, furunculosis, the pyodermatitides in general, etc.

The possibility that a nervous influence by itself alone may give rise to erythema is demonstrated by *emotional erythema*, usually very transitory, produced by emotions such as shame, anger, joy, etc., on the face, the ears, the neck and sometimes the upper part of the chest (*erythema a pudore*). Abnormal psychic or nervous conditions, such as are met with at the time of the menopause, in exophthalmic goitre, etc., exaggerate this tendency to blushing, which in some patients is almost a real infirmity.

The existence of *reflex erythema* of gastro-intestinal, urethral, uterine origin, etc., has also been admitted and it is naturally difficult in such cases to distinguish between the role of the intoxication and the infection.

Pathogenesis.—The pathogenetic mechanism is certainly not uniform in the group of erythema. Physiology teaches that hyperemia is subject to vasoconstrictor and vasodilator mechanisms located in the bulbo-medullary center and at the periphery. These may become involved in a very variable manner; by a local poison, in the case of a flea-bite; by a psychic disturbance in emotional erythema. Difficulties arise in such common cases as when an individual is attacked by erythema, for instance, after eating mussels; the question is whether to interpret the erythema as directly toxic, autotoxic through indigestion or of reflex nervous origin. Physicians used to hesitate between these theories and with good reason, for they are probably all wrong. In a patient in my service, who was attacked by urticaria under these conditions, the existence of

anaphylaxis was demonstrated by Ch. Flandin and Tzanck. At any rate, it is well to keep in mind that a series of experimental investigations, in harmony with the findings of pathological histology seem to prove that in the great majority of erythemas, those at least which are not absolutely transitory, the process involved is a local inflammation and not simply an angioneurosis, as had been assumed.

In some instances of *infectious erythema*, the actual presence of microbes has been recognized in the skin. This is the case in the rose spots of typhoid fever, some forms of gonorrheal and pyococcal erythema and syphilitic roseola. When no microbes are found, it may be assumed that they have already disappeared. But it is extremely probable that in numerous cases the microörganisms act merely indirectly.

Recently acquired knowledge of *serotherapeutic erythema* sheds some light on the pathogenesis of erythema in general. I shall have occasion, further on, to point out that the complication of serum-treatment must be interpreted as manifestations of anaphylaxis. It is probably not the anaphylactic poison itself, the apotoxine of Ch. Richet, which acts in the serum-eruptions. There is good reason to believe that changes in the composition of the blood are responsible, perhaps the presence of precipitins, as assumed by Hamburger and Moro, Marfan and Rovere, or perhaps other antibodies or derived substances of local origin, or disseminated by embolism.

What is true for the serum eruptions is equally applicable perhaps to toxinic erythema and infectious erythema. We know that the injection of Koch's tuberculin into tuberculous subjects invariably produces a local erythematous patch, sometimes of erysipeloid type; in some cases it causes, moreover, the appearance of a generalized eruption of more or less urticarial erythema. It is probable that in the last named cases the composition of the blood is different.

In children suffering from severe diphtheria and treated with serum, there occurs not only an attack of urticaria or marginate erythema, but scarlatiniform and morbilliform erythema is likewise observed and the question arises if these eruptions are referable to the serum or are of infectious origin.

The subject is complicated and not yet settled. What seems to be established is that in the pathogenesis of many forms of erythema, just as is true for certain urticarias, purpuras, etc., there is reason to admit the intermediation, between the pathogenic agent (foreign serum, microbe, toxine) and the eruption, of some change in the humoral composition of the blood.

The ancient formulas in regard to the pathogenic factors of erythema are thus peculiarly vindicated. I was right in asserting that "while certain agents more or less readily produce eruptions in all persons, others necessarily require a predisposition and active co-

operation of the organism." E. Besnier correctly taught that "the apparent cause of the eruption sometimes seems to determine neither its form nor its course;" and that "the ordinary causes merely bring out the morbid tendency." It is thus understood why Brocq refused to group erythema among the true morbid entities, but made it one of the chief types of his "cutaneous reactions." Recent investigations serve to confirm these clinical observations.

From all that has been said so far, it results that the few *dermatoses of erythematous type*, to be presently described, cannot be referred to a definite etiology and pathogenesis. It would be a much too simple as well as an erroneous conception of the subject to assume outright that *intertrigo* is an erythema of mechanical origin; that *sunburn*, *frost-bite*, and *livedo* are dependent on a physical cause; that *rosacea* results from an auto-intoxication; or that *erythema multiforme* is infectious. These are not morbid entities, but simple *syndromes*.

Syphilitic roseola is the only example I shall quote of an erythema having a definite cause, the eruptive manifestation of an actual disease.

INTERTRIGO.

The congestive redness which seems to result from mutual friction of two contiguous surfaces, is designated as *intertrigo*. This term should preferably be employed only as an adjective, attaching it to the name of the dermatosis produced by these conditions. As a matter of fact, aside from *intertrigo-erythema*, there exists *intertrigo-eczema*, etc.

Intertrigo-erythema is ordinarily observed especially in obese individuals, in the fold between the buttocks and on the internal surface of the thighs, for example after a lengthy march. In stout women, it is also seen under the breasts, in the hypogastric fold, in the groin and axillary regions, or in newborn infants, on the buttocks, the folds of the neck, etc.

The more or less vivid redness is bounded by irregular or diffuse margins; there is local heat or pruritus; pigmentation and sometimes lichenization finally develop. To the mechanical causes are added the harmful effects of sweating, maceration, regional secretions, secondary fermentations and infections; so that the erythema is frequently complicated by dermatitis, lymphangitis, pyodermatitis, eczematous changes, etc., which may spread.

The inguinal intertrigo of adults is distinguished from erythrasma (p. 532) and from the eczema marginatum of Hebra (p. 524) by its always symmetrically affecting the two sides of the fold, which is often fissured; and especially by the lesions of intertrigo being

neither uniform, nor polycyclic, nor marginate, having on the contrary more or less diffuse borders.

Infantile gluteal erythema is related to intertrigo and radiates from the intergluteal fold to the thighs, the back, the abdomen, even as far as the heels. It is extremely common and due not so much to friction as to contact of the skin with the dejecta, especially in the presence of diarrhea or athrepsia. Sometimes, the condition is a simple hyperemia, often of a coppery red color, covering a large area, or in patches; in other cases, the erythema is complicated by fissures or vesicles and oozing erosions, briefly by true eczematous changes (intertrigo eczema of the newborn) as shown by Marcel Ferrand; or again, it becomes covered by ulcerations and pyodermal lesions.

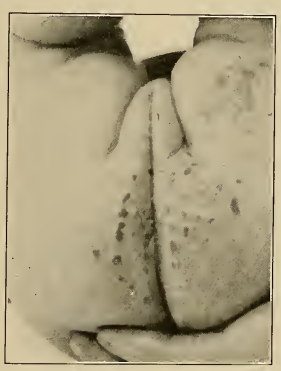


Fig. 1.—Syphiloid (non-syphilitic) dermatitis of the buttocks in the newborn.

In a form described under the name of *papulo-lenticular erythema* or *posterosive syphiloid* by Sivestre and Jacquet, the erosions appear as raised, moist papules.

Several varieties of these eruptions very closely simulate the polymorphous syphilides of the newborn, as may be seen from the illustration (Fig. 1) and the description of the latter. The diagnosis, in doubtful cases, rests on the general examination of the child and its environment, the direct discovery of the spirochete in the lesions, the Wassermann reaction of the little patient and its mother and finally on the course. As long as any doubt remains all precautionary measures must be taken to guard against contagion.

The *treatment* of intertrigo consists in the frequent application of astringent or weakly antiseptic soothing lotions. An essential precaution is the successful separation of the affected surfaces by

means of sterilized gauze, or especially by neutral mineral powders. Zinc oxide pastes are also appropriate, but salves are often injurious. The diet of children must be closely watched, and, if necessary, corrected.

ERYTHEMA SOLARE SEU ACTINICUM.

The inflammatory redness which follows after exposure to the rays of the sun is known as *sunburn*. At the end of a few hours, the exposed surfaces, usually the face, especially the nose and ears and the hands, present an intense congestion, with swelling, burning and itching; this lasts a few days, then the epidermis desquamates in large shreds and only a slight pigmentation is left behind. Not all individuals exposed to light radiations are affected to the same degree. Delicate skins, blondes, certain neuropaths and persons not accustomed to the open air, are more susceptible to sunburn, especially in the spring near the water, or in excursions on glaciers.

Insolation, or *heat-stroke*, is an altogether different accident, sometimes very grave, characterized by general phenomena instead of cutaneous lesions and referable to a different pathogenesis.

Solar erythema differs also from burns. It has been more than sufficiently demonstrated that it is not the caloric rays, but the chemical rays of the spectrum, the violet and ultraviolet rays which are responsible in these cases. The electric arc-light is accordingly capable of producing an entirely similar *electric erythema*. The same chemical radiations are utilized in *phototherapy*, for example in Finsen's method.

A few very simple precautions, such as wearing a veil, the application of creams, lotions, or powders made with quinin or esculine, suffice to protect against sunburn. The treatment is that of an artificial dermatitis or a burn of the first degree. Numerous observers believe, not without good reason, that the action of the solar rays plays a part in the production of *pellagrous erythema*.

Pellagra is a non-contagious general disease, endemic in many countries, notably in Italy (mal della rosa), where for a long time it has constituted a veritable scourge; in the Balkan provinces, in Spain, in southwestern France, in Egypt and in Asia minor. In the last ten years, important foci have been discovered in the United States, Guyana, the Antilles, and a few cases in Great Britain.

Pellagra usually starts in the spring, followed by seasonal outbreaks. The *erythema* which often betrays its presence, manifests itself on the exposed parts, especially on the back of the hands and the wrists, sometimes on the face, neck, upper part of the trunk, and even on the dorsal aspect of the feet. It consists of a sombre reddening, with more or less clearly marked outlines, sometimes complicated by small bloody suffusions, fissures, or bullæ. [The sharp

line of demarkation between the affected areas and the normal skin is very characteristic of pellagrous erythema.] At the end of two or three weeks the skin becomes pigmented, darkens, and is shed in large shreds, finally undergoing atrophy, so as to resemble the skin in certain senile cachexias. *Lesions of the mucous membranes* are not infrequently observed, for example a bullous and later on diphtheroid stomatitis, as well as an analogous vulvitis.

Aside from the cutaneous symptoms, a very marked loss of strength is noted after the onset of the disease, followed by a true asthenia, with serious digestive disturbances, anorexia, fetid diarrhea, emaciation, sometimes chills and fever, ordinarily serious

FIG. 2.—Pellagra. Siler, Garrison and MacNeal, Thompson-McFadden Pellagra Commission.

mental and nervous disturbances, paresthesias, hyperesthesias, convulsions, melancholia and delirium with a tendency to suicide. Many patients drift into insane asylums. The mortality in certain foci is appalling, but there are numerous abortive cases which may remain unrecognized.

The theory according to which pellagra is due to a chronic intoxication by spoiled maize (Zëism) has been widely accepted. It is claimed, however, that pellagra appears also in regions where maize (Indian corn) is never used. The infectious theory, which is now opposed to the maize theory, acquires much probability from recent epidemiological investigations, notably those of L. W. Sambon: Simulides or other insects are assumed to act as carriers of a special

protozoan parasite, supposed to be the causative agent of pellagra. There would thus be a remarkable parallelism between this disease and malaria. [The etiology of pellagra is not clear but the weight of evidence at present would indicate that the disease is one of malnutrition due to a defective diet. The parallelism is rather with beriberi than malaria.] Doubt prevails as to the nature of an absolutely identical or sometimes attenuated syndrome which may be observed in European countries among cachectic individuals, inebriates, or in those in a state of physical and moral deterioration. Some interpret this syndrome as a *pellagroid erythema* of alimentary or cachectic origin, whereas others admit no essential difference between this pellagroid and true pellagra. The point has not yet been settled.

ERYTHEMA PERNIO OR FROST-BITE.

Everybody knows frost-bite, in the form of a purplish and painful reddening frequently observed during the cold season, especially in children and youthful individuals. In order of frequency, these lesions affect the hands, notably the ulnar border and the fingers, the toes and heels, the ears, the nose and more rarely the cheeks.

In the *first degree* they consist of a swelling of the skin, which is of a dark or bluish-red color, tense, glazed, indurated and cold to the touch. Although the erythema is evidently due to stagnation, there occurs an instantaneous active hyperemia when the parts are too rapidly heated, the sensation of stiffness changing into an itching and very painful burning sensation.

The erythema may be limited to patches, or diffusely outlined or even generalized over nearly the entire region.

In the *second degree*, the frost-bite is "open," as it is ordinarily described. The ulcerations result either from cracks which appear in the folds, or from sometimes rather large bullæ which originate on the swollen surfaces, probably under the influence of infection by pus-cocci. A persistent exudation follows, or a suppuration with crusts, which may lead to spongy bleeding ulcers, causing an actual loss of substance. Under these conditions, manual labor or walking sometimes become impossible.

Untreated frost-bites usually last with exacerbations through the entire winter and disappear in the spring; but there are exceptional cases which persist even in the summer.

The part played by the season and the local action of the cold are beyond dispute. However, the determining causes must not be allowed to overshadow the predominating importance of the soil. The age of five to fifteen years predisposes to frost-bite; but not all children are attacked, and moreover, cases are observed in young and adult individuals. Anemia, the so-called lymphatic constitu-

tion and arterial hypotension, are evidently very often involved; it may even be stated that the frequently observed association of frost-bite with habitual acro-asphyxia, with adenopathies, with the scrofulous diathesis, might cause them to be classified among the manifestations of attenuated tuberculosis which are now designated as tuberculides (p. 563). This view is supported by the not uncommon cases where the frost-bite is apparently transformed into erythematous lupus, the chilblain lupus of Hutchinson; indicating a probable relationship between these two affections, the second of which is considered as a tuberculide (p. 571). The cases in which chilblains give rise to angiokeratoma, or alternate with papulo-neurotic tuberculide, point in the same direction.

Treatment.—In frost-bite of the first degree, the patient's sufferings are greatly relieved by means of lotions made with lukewarm or warm water, followed by rubbing with camphorated spirits, alcohol with a little iodin, or an infusion of tannic acid. Salves or powders made with tar or ichthyol are sometimes useful.

In case of cracks or ulcerations, a moist occlusive dressing is applied, or applications of linimentum calcis or vasolanoline, until cicatrization is obtained, before resorting to astringents and keratoplastic agents, such as ichthyol, resorcinol, etc.

Hydrogen peroxide in local baths of fifteen to twenty minutes, repeated two or three times daily, is often remarkably successful.

The "biokinetic method" of Jacquet, provided it is properly applied, constitutes a preventive and excellent treatment of frost-bite and chilblains. The patient must actively move (8 or 10 times daily for five minutes) all the joints of the affected extremities, which meanwhile are kept in an elevated position.

General medicinal treatment, as employed in the lymphatic and scrofulous diathesis, must not be omitted, in the form of fresh air, dry or alcoholic rubs, cod-liver oil, arsenic, calcium, iodide of iron, etc. Certain forms of opotherapy are sometimes indicated (suprarenal extract, etc.).

The disease known as *trench-foot* (said to be frozen feet), and frequently observed in the great war, differs from chilblains in many respects. It occurs in soldiers who have been obliged to stand several days in cold water or mud, even in the absence of ice, and is the result of the enormous withdrawal of heat, together with the effects of the [prolonged erect or] slanting position, the general fatigue and sometimes the constriction of the legs and feet. It consists of a purplish and very painful swelling, which interferes with walking, either without cutaneous lesions or accompanied by purpura, bullæ, or even more or less extensive and deep sloughs. In such cases we have noted, with Civatte, a very persistent, painful anesthesia of the distal end of the foot, indicative of peripheral

neuritis. Vascular lesions are held responsible by others. The duration may be several months, even in cases which receive proper treatment with baths or douches of hot air and kinetotherapy in the elevated position.

ACRO ASPHYXIA AND LIVEDO.

Acro-asphyxia and livedo are two other forms of passive erythema which must be considered in connection with chilblains.

Acro-asphyxia is a chronic congestion of the extremities, which are of a purplish red color, habitually cold and often damp and flaccid. The ischemic spot produced by digital pressure requires a long time, sometimes nearly a minute, to resume its red color by the inflow of blood.

This affection is observed under a variety of conditions. Obviously, this syndrome can be produced by a spasm of the veins, a change of the venous walls or a lowering of the arterial tension (cardiac or pulmonary lesions, cachexia). Permanent nervous disturbances, nutritional impairment and chronic exogenous or autogenous intoxications may undoubtedly lead to the same result. Children or youthful individuals having the scrofulous diathesis are particularly susceptible. The manifestations are accentuated by cold.

Without itself causing serious inconvenience, acro-asphyxia creates a seat of predilection for chilblains, tuberculides, angiokeratoma, artificial dermatitides and pyodermatitides and imposes a sluggish, dragging tendency on the course of all these skin lesions.

Livedo (livedo annularis a frigore or reticular asphyxia) has a practically identical etiology and significance. It is observed especially on the outer aspect of the forearms, arms and thighs, on the flanks and sometimes over almost the entire integument.

It consists of a persistent purplish reddening, increased by cold, which appears on the skin as a network made up of strands of variable size, enclosing round or oval meshes. These meshes have a normal color and correspond to the territories of direct blood supply, whereas the strands of the network represent the anastomotic zones between these territories. Livedo accordingly represents the "negative" of a roseolar eruption.

ROSACEA.

Rosacea (acne rosacea, acne rosea, gutta rosea, Fr. couperose) is a special affection of the face, a passive erythema, persistent but variable, sometimes complicated by pustules. Its principal localization is the nose, the cheek and the middle of the forehead; by extension, it may reach the chin and the temples.

Etiology.—Rosacea does not occur in children; it appears either after puberty, or more generally toward the age of forty or fifty years in women at the time of the menopause. It usually disappears in old age.

Many different factors may enter into its etiology. It is very frequently preceded by a stage of acute congestion of the face, recurring in attacks and designated as *erythrosis facialis;* on the other hand, it almost invariably enters into the symptom-complex of *kerosis* (p. 196) with or without seborrhea. Both these morbid conditions act as intermediaries between the general disturbances presently to be enumerated and the chronic erythema of the face which we call rosacea.

Most commonly, digestive disturbances are responsible, such as habitual constipation, gastro-intestinal fermentations, hepatic dyspepsia, abuse of stimulants, etc. Many people, especially young women, are subject to attacks of acute congestion of the face, diffuse or in patches, after eating too rapidly without sufficient chewing(tachyphagia), or under the influence of certain foods or beverages. This facial erythrosis, which seems to be the effect of a reflex of gastric origin (L. Jacquet) may recur during several years, or it may change rather rapidly into rosacea. In other cases, rosacea is established from the start with persistent red spots which have a tendency to become confluent. This is apt to be the case in inebriates, especially wine-drinkers; the florid complexion and red nose are not a sign of splendid health, but rather an index of gastro-hepatic dyspepsia.

Disturbances of the genital functions, especially utero-ovarian; the menopause; dysmenorrhea; inflammations of the tubes and ovaries; metritis, etc., likewise predispose to rosacea and especially to its localization on the chin. Chronic cardiac and pulmonary lesions are occasionally responsible. Affections of the nasal fossæ and sinuses apparently invite the skin lesion in a number of cases and the same is true for dental caries, which through the loss of many teeth, moreover, becomes an important cause of dyspepsia.

The action of cold, heat, or wind; the menstrual periods; cutaneous irritations of all kinds, contribute to the renewal of attacks and to the coalescence of the red spots, which become darker, of a purplish or dark blue color, or to the exacerbation of rosacea already established.

Symptoms.—In its fully developed stage, rosacea consists of a persistent more or less diffuse redness, without elevation of the local temperature. After a while, superficial telangiectases make their appearance; small undulating veins ramify over the nasogenial grooves, furrow the wings of the nose and twist in intertwining twigs over the nose, the cheeks, the temples, the forehead, and the

chin. In well-marked cases of kerosis, the skin becomes swollen, thickened as a whole, and a few hypertrophied sebaceous glands may project over the surface. This condition in particular is designated popularly as "gin-blossom."

Although rosacea may run its course unmixed, it very frequently becomes complicated by folliculitis, especially in patients having seborrhea. The majority of authors have on the other hand interpreted this acneiform folliculitis as primary and causative of the surface redness. Careful observation of these patients shows this to be incorrect. These follicular inflammations have also been generally confused with the papulopustules of acne vulgaris (p. 385). Unna has shown that they differ from the latter by the absence of comedones, by their superficial situation, by their localization and by the age of the patients.

The follicular inflammations of rosacea appear in attacks affecting from two to ten follicles at once; they pass through all the stages of papules, pustules, crusts, lasting from two to four days and are constantly repeated. Overindulgence in food, however, may give rise to a crop of twenty to thirty inflamed follicles. They aggravate the previous condition, unpleasantly affecting the patient's appearance and inevitably leaving at least a minute cicatrix. An extreme degree of rosacea seriously disfigures the sufferer. The purple pimply nose, deformed by swollen ridges separated by deep grooves, traversed by large dilated veins, is increased in size in all directions; this condition has been described as a separate affection under the name of rhinophyma (p. 374). The other parts of the face are sometimes likewise purplish, swollen and scattered over with pustules, cicatrices and telangiectases. The general impression is far from esthetic.

Diagnosis.—The diagnosis of rosacea is generally easy, except perhaps when it presents certain features suggestive of iodides and bromides, or of lupus pernio, which is by no means limited to the nose, or of lupus erythematodes, which is characterized by the sharp outline of its margins. The differentiation from certain tertiary syphilides of the middle of the face may prove extremely difficult.

Treatment.—In the first place, the various etiological factors referred to above must be looked for and appropriate diet and internal treatment prescribed. The local treatment varies with the degree of the affection and the presence or absence of complications. In a moderately severe or mild case, the rule is to exhaust the power of topical agents before resorting to the obliteration of the dilated vessels. Ointments are often badly tolerated. Sulphur and ichthyol pastes, powders, bathing with lukewarm naphthol or ichthyol soap solution, sprays or warm lotions with astringent fluids with or with-

out the addition of bichloride are decidedly preferable. A sulphur and camphor lotion may by itself alone lead to considerable improvements provided the general indications have been met. Local massage, or better still, frequent sessions of facial gymnastics, advocated by L. Jacquet are often very efficient. Finally, when required, the treatment may proceed to obliteration of the telangiectases, preferably with the galvanocautery, or exceptionally by means of electrolysis or scarifications.

In a grave case of rhinophyma, after relieving the irritation and checking the suppuration by the application of mild antiseptic sprays and dressings, surgical intervention may prove necessary in the form of nasal decortication.

ERYTHEMA MULTIFORME.

Under the name of multiform or polymorphous exudative erythema, Hebra distinguishes in the heterogeneous group of erythema a syndrome which one is almost tempted to regard as a disease. In its behavior it approaches the eruptive fevers, but as it is neither specific nor contagious, it must be described as pseudo-exanthematic.

Symptoms.—The eruption, which is often accompanied by systemic phenomena, consists either of erythematous papular elements, or of vesicles or bullæ, or finally of nodosities. It is somewhat uncommon to see these different forms of elements combined in the same individual.

The following types may therefore be described: *Papulo-erythematous*, which alone will be discussed here; a *bullous* type or *hydroa* (p. 175) and the so-called *erythema nodosum* (p. 265). Polymorphous erythema of the papulo-erythematous type is characterized by nummular or lenticular congestive spots, the center of which promptly becomes cyanotic; these spots spread, remaining flat or becoming wheal-like or papular, sometimes discoid, or depressed in their center (Fig. 2). The livid color of the spots, their bright-red border, their manner of development and the distribution of the eruption are sufficiently characteristic. The lesions may also be whitish in their middle, appear to be bullous or actually become so in some cases; or again, their center may become purpuric. Their extension is rapid and sometimes gives rise to marginate spots, or even to rings, through the obliteration of the central area (erythema annulare).

The *eruption* is composed of a very variable number of elements, disseminated or arranged in groups, sometimes confluent, usually symmetrical. The seat of predilection is the dorsal surface of the wrists, hands and forearms; it sometimes occurs on the fingers, the

elbows, the nape of the neck, the forehead, the knees, and rarely the feet. Heat, pruritus and local tension are present, often regional swelling, sometimes arthralgias or even arthritis simulating acute articular rheumatism; prostration, headache, gastric disturbance and some fever at the onset. As a rule the eruption extends by successive attacks. The entire duration is from one to five weeks. Moderate desquamation is noted on its subsidence.

Relapses are not uncommon; and Brocq finds in these relapses a connecting link between the recurrent cases with grouped lesions accompanied or not by especially painful phenomena and the painful polymorphous dermatitides (p. 179).

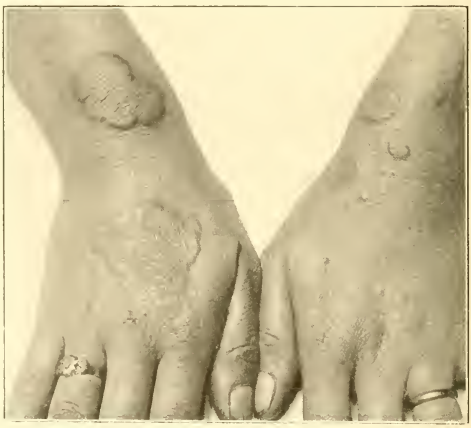


FIG. 3.—Polymorphous erythema of the erythematous papular type.

Etiology.—Notwithstanding its appearance of a fairly well defined morbid entity, polymorphous erythema seems due to quite a number of ordinary causes. The effects of cold, alimentary or medicinal intoxications; infections, such as rheumatism, the anginas, gonorrhea, syphilis, tuberculosis, leprosy and perhaps reflex actions or auto-intoxications may all be present, without lending a special feature to the disease. In other words, the true cause is not known. It is probably a reaction of the organism under the influence of various intoxications or toxins. [There is reason to believe that low-grade infections are the most frequent etiological factor.]

Treatment.—In the first place this must not be irritating. According to the cases, purgatives, rest, a light diet may suffice; calcium chloride, aspirin and salicylates are often useful; iodides have been

highly recommended, but are rather injurious. Locally, applications of neutral powders will suffice, with protective dressings if needed.

As it has been shown by the work of Prof. Landouzy and his pupils that polymorphous erythema in a considerable number of cases is due to tuberculosis, it is advisable to prescribe an appropriate hygiene for convalescent patients and to keep them under prolonged observation.

SYPHILITIC ROSEOLA.

No other infectious erythema possesses the same importance as syphilitic roseola; which is accordingly selected for a special description. It is the most common of the cutaneous syphilides and consists of an eruption of spots, at first of a peach blossom color, of a deeper pink after a few days, of nummular size, rounded or oval shape and indefinite margins; these spots being at no time either squamous or pruritic. This eruption is scattered indiscriminately on the flanks, the chest, the back and the abdomen, sometimes extending to the neck, the limbs as far as the palms and soles, very rarely to the face where it is observed only on the forehead.

In the absence of early and radical treatment, it usually appears from forty to fifty days after the chancre, beginning on the flanks and hypochondriac regions, developing in a fortnight and lasting from three to six weeks or two months. It occasionally fails to make its appearance even in cases where the chancre has remained unrecognized and no treatment has been instituted. Not infrequently it escapes the patient's attention and must be looked for. Sometimes it is so profuse and high-colored as to give the skin a mottled appearance.

The roseolar elements, especially when small, of lenticular dimensions, may cause a slight protuberance, as the result of some congestive edema; representing *urticarial roseola.* The form in which the macules develop into papular syphilides is designated as *papular roseola.*

There may be a recurrence of the roseola in the course of the first or second year, or even later. These *recurrent roseolas* are usually rather pale and composed of larger and less numerous spots, which are often circinate or annular; their duration is apt to be prolonged.

Tertiary roseola, or *tertiary erythema,* is the name given to dull red. non-squamous and non-infiltrated spots, polycyclic or circinate, occupying the trunk and limbs and very rebellious to treatment. These lesions are met with in long-standing syphilitic cases which have been subjected to energetic and prolonged treatment. They may be interpreted as the attenuated equivalents of tuberculo-ulcerative syphilides.

Syphilitic roseola differs from the *pityriasis rosea* of Gibert and

from *eczema* by the absence of desquamation of any kind; from the *balsamic roseolas*, by its less intense color, its slow development and the absence of itching. Among the affections capable of causing real diagnostic difficulties may be mentioned the *roseola of leprosy* and the *roseola of mycosis fungoides*, but these are very rare and are accompanied by other symptoms of these two diseases. *Antipyrin roseola* is likewise objectively identical, but is the least common of antipyrin eruptions and lasts only eight to ten days. The most ordinary practical difficulty results from the *macules* sometimes remaining on the skin after a pyodermatitis, scabies, or pediculosis. All doubts will be cleared up by the history and the topographical distribution of the lesions.

Diagnosis.—The diagnosis of syphilitic roseola should always be carefully confirmed through the demonstration of other signs of the infection, such as glandular enlargement, mucous patches, remains of the chancre, alopecia, headache, etc., or by means of the Wassermann reaction.

Treatment.—Treatment should be prescribed only when the diagnosis is positive; specific medication suffices, whereas irritating topical applications, sulphur baths and all methods which congest the skin, usually merely aggravate and prolong the eruption.

CHAPTER II.

URTICARIA.

The name urticaria is applied to an eruption composed of peculiar elements which for lack of a special denomination are described as *urticarial patches* or *papules* or *wheals*. This eruption is essentially pruritic in character.

The eruptive element of urticaria is an elevated distinctly outlined efflorescence, light pink in color or of an opalescent white with a pinkish areola; of a rounded or oval shape, sometimes polycyclic and of solid consistence. Its dimensions, usually nummular, vary from the size of a lentil to that of a more or less extensive surface.

The *eruption* consists of an extremely variable number of elements, appears suddenly, in a few seconds, is transitory or ephemeral, vanishing after a few minutes or hours; the pinkish color fades, the elevation flattens out and, with some exceptions, no trace is left behind.

The eruptive element is typical, but in spite of the characteristic eruption, urticaria can in no way be regarded as a disease. It is often a simple *symptom*, a cutaneous reaction which may be provoked by a great variety of causes. It is sometimes a *syndrome*, when the eruption assumes the behavior of a pseudo-exanthematic eruption and is accompanied by general disturbances; but it is not a disease in itself.

It cannot be overemphasized that a fundamental characteristic of urticaria is that it is invariably associated with severe itching, heat or formication, so that scratching becomes imperative. The *pruritus* often precedes the appearance of the efflorescences; it is usually more diffuse than the eruption.

In an attack of urticaria, the skin of the affected regions presents almost invariably, at any rate temporarily, a congestive tendency which may be designated as *urticarism;* owing to which new lesions may be provoked by scratching, rubbing, the action of cold or irritation of any kind. Jacquet has shown that no new lesions appear under really occlusive padded dressings. It may accordingly be stated that in urticaria the *pruritus is primary* as compared to the eruption. Many dermatologists therefore group urticaria under the heading of *pruritus*.

The *site* of urticaria is extremely variable; it is localized or regional or it may be generalized; it affects preferably the trunk and

limbs, but sometimes also the palmar and plantar regions, the face and the hairy scalp. In regions with a loose cellular tissue, such as the eyelids, the prepuce [the lips], etc., the eruption manifests itself in the form of an enormous *urticarial edema*, with diffuse margins, rather alarming, but transitory.

Fig. 4.—Acute urticarial eruption, on the flank of an adult man (developing in the course of axillary pyodermatitis).

The *mucous membranes* may be invaded, notably the mouth, pharynx, and larynx; redness and edema occur, and the latter may interfere with respiration. Mention has even been made of urticaria of the nasal fossæ, the bronchi and the digestive apparatus. This is imaginary and merely a theoretical explanation of hay-fever, certain forms of asthma and of paroxysmal diarrhea.

The general phenomena which may accompany the onset of certain very severe attacks of urticaria consist in fever, sometimes high but very transitory (*urticarial fever*) and prostration, with more or less marked digestive disturbances.

Varieties.—The varieties of urticaria are numerous and are derived from the morphological aspect of the lesions or the course of the eruption. The configuration of the eruptive lesions, discoid, annular, circinate or linear; as well as their porcelain tint, have no special importance. However, the center of the spots may assume a purplish hue, resisting digital pressure; this is *hemorrhagic urticaria*, separated merely by a shade from *purpura urticans*. The bloody infiltration in the skin in these cases undergoes the usual transformation into pigment, so that the elevations leave brownish macules behind; this *pigmented urticaria* must be carefully distinguished from *urticaria pigmentosa* (p. 703).

The appearance of a small, hard and persistent papule in the center

of the urticarial spots characterizes the *papular urticaria* of several authors. This is described in this book under the name of *strophulus* (p. 141).

In certain very unusual cases, the urticarial elevations become topped by a blister containing a serous and later a purulent fluid which dries in crusts. This *bullous urticaria* is not readily distinguished from certain forms of pemphigus (p. 175). [Both papular and bullous urticaria are not uncommon in the urticarias of infants and children.]

The eruptions which have sometimes been described as *urticaria perstans*, the lesions of which persist for months or years, and which must not be confused with constantly recurring chronic urticaria, are probably related either to the prurigos (p. 498) or to the pre-mycotic eruptions (p. 657).

Giant urticaria is a separate clinical form, which will be briefly discussed further on. The same remark applies to *factitious urticaria.*

The behavior of urticaria permits the distinction of an *accidental form*, instantaneously produced by an external irritant; an *acute form*, in which the eruption consists of a single attack, or a few successive or overlapping attacks, so that the patient is well again in twenty-four hours, in two or three days, or in a week; and a *chronic urticaria*, in which successive attacks occur continuously, or at intervals, during months or even years. In the last named cases the itching becomes a real torment, disturbing sleep and exhausting the patient, as a result of the incessant scratching, the skin becomes covered with excoriations, crusts, and pigmentations. The condition may terminate, according to the age of the patient, in Hebra's prurigo, or in ordinary diffuse prurigo (pp. 494 and 496).

Etiology.—The causes of urticaria are in part local, direct or determining; and in part general, indirect or predisposing. They may become associated in variable proportion in producing the eruption.

It will be readily understood that a very active direct cause may by itself alone give rise to urticarial efflorescences, in all persons indiscriminately; on the other hand, in very strongly predisposed individuals, the most ordinary and trifling irritant may produce the eruption. Between these two extremes all variations are met with.

Authors who maintain that urticaria should be considered as a disease having a deep-seated cause in the organism, refuse to include with it such artificial efflorescences as that produced, for instance, by the stinging nettle, although the very name of the disease is derived from that plant [urtica]. This conception has certain arguments in its favor, but I cannot accept it for the reason that the eruption is identical in urticaria of external and of internal origin and that it is impossible to draw a sharp line between these two affections. All

urticarial eruptions are therefore grouped together in this book under the heading of urticaria.

External Causes.—Stings produced by the glandular hairs of nettles, the stings of mosquitoes, bed-bugs and fleas, contact with the hairs of processional caterpillars, with medusæ, or various poisonous plants, give rise after a few seconds to severe pruritus and slight reddening. Next, especially on scratching, or when the congested and sweaty skin is washed with cold water, an eruption of *accidental* urticaria, or even of *acute* generalized urticaria in predisposed individuals, occurs. A local cause should therefore be looked for in the first place in cases of urticaria as in any pruritus.

It is superfluous to enumerate all irritants capable of inducing the eruption in temporarily or permanently susceptible individuals. The slightest friction or contact with water or even air may be sufficient.

Internal Causes.—Clinical studies of the manifold conditions under which urticaria is observed, has led to the following conclusions: The predisposing factor in urticaria was supposed to be referable to somewhat indefinite nervous states, often of congenital origin in children and youthful individuals, of acquired origin in adults, due to mental strain, hysteria, neurasthenia, chronic intoxications, or weakening from any disease. Violent emotions, anger, or fear, may precipitate a crisis.

Digestive disturbances play the principal part in other cases, especially gastric dyspepsia, dilatation of the stomach, diseases of the liver, habitual constipation. An attack of indigestion, or partaking of certain foods or drinks, brings on the eruption in some individuals, the following substances appearing particularly injurious; Deep-sea fish, crustaceans, shellfish, especially mussels, pork, game, eggs, preserves, cheese, ices, strawberries, raspberries, wines, tea and coffee, many medicinal agents, etc.

In *urticaria ab ingestis*, a reflex action of gustatory or gastric origin has sometimes been assumed on account of the very brief period before the onset of the attack. The reflex may perhaps also start from the genito-urinary organs, according to the older views.

The urticaria from abnormal intestinal fermentations, from renal or hepatic insufficiency, from gout, pregnancy, etc., was interpreted as *autotoxic*, while the urticarial eruption which sometimes precedes or accompanies the eruptive fevers, intermittent fever, etc., was considered as *infectious* urticaria.

It was known finally that several varieties of urticaria may occur in blood diseases, leukemias, etc.

In the last few years, however, the subject has taken on another aspect through laboratory and experimental investigations along the line of *anaphylaxis* (p. 460), as will be discussed further on.

The fact has now been established that the majority of urticarias are due to anaphylaxis.

Pathogenesis.—The eruption of urticaria obviously results from a local congestion of the cutaneous vessels with serous exudation especially in the papillary body, sometimes extending to the hypoderm and more rarely to the epidermis. The firmness of the urticarial wheal, its pallor resulting from the compression of the bloodvessels and the cleavage of the epidermis in the bullous form are proof that the plasma is exuded under high pressure.

It should be kept in mind that toxic or infectious erythema may be urticarial, so that no sharp line can be said to exist between the two eruptive types of erythema and urticaria. Histology furnishes no data to explain the phenomenon of urtication. In simple primary urticaria, an excised segment usually presents no lesion, provided the blood-pressure and edema have disappeared; sometimes, a slight local polynucleosis is noted. [Gilchrist has found distinct evidences of inflammatory changes even in the most recent wheals.]

As to the pathogenic mechanism, Török and Vas have shown that the fluid exudate in urticaria contains more albumin than that of the mechanical edemas and is analogous to that of the inflammatory exudates. The inflammatory or angioneurotic character of the urticarias has been discussed by several foreign authors. Török and Philippson successfully produced experimental urticaria in dogs, by inserting into the cutis capillary tubes filled with various substances among which may be quoted: peptones, pepsin, trypsin, cadaverin, putrescin, morphin, atropin, antipyrin, antidiphtheritic toxin, staphylococcus toxin, etc.; hot water, formic, oxalic and uric acids, syntonin, casein, etc., are said to be less active; while glycocoll, asparagin, purin derivatives, bilirubin, etc., are apparently inactive. Possibly some of these substances which act in concentration also play a part when they circulate in even infinitesimal dilution in the blood.

More widely applicable and more conclusive are the experiments which have demonstrated the anaphylactic character of the majority of urticarias. For those which are produced by the sera (page 477), proof has been furnished by Arthus, Theobald Smith and others. Reasoning by analogy, the same mechanism has been invoked in all cases where injection or absorption of a foreign albumin enters into consideration. Widal, in collaboration with Abrami, Brissaud and Joltrain, has shown that in an attack of alimentary urticaria, the cutaneous phenomena are preceded by a set of blood and vascular phenomena, an actual "hemoclastic crisis," identical with that of anaphylactic shock; a fall of blood-pressure and a rapid leukopenia being its essential features. Moreover, the transmission of passive anaphylaxis to guinea-pigs has been

successfully carried out by Bruck, for urticaria due to pork, and for the eruption following mussels, by Flandin and Tzanck working in my laboratory. The watery fluid of hydatid cysts, which causes urticaria and sometimes grave symptoms when effused into serous cavities, although it is not poisonous has been shown to produce anaphylaxis, by Chauffard, Boidin, Laroche and Devé.

Thus it becomes extremely probable that under a large number of conditions, the susceptibility to urticaria consists in an anaphylactic state and that the determining cause merely liberates the eruption.

Treatment.—In the first place, it is necessary to determine that the urticaria does not depend upon an external cause, parasitic or other. Acute attacks are suggestive of some article of food; and a purgative and a strict diet based on the list of presumably harmful substances (see Therapeutic Notes), with some local applications may constitute sufficient treatment.

In the case of chronic urticaria, it behooves the physician to investigate with care the general disturbances so as to remedy these. Thorough hygiene of the nervous system and a strict alimentary regimen, sometimes a closely prescribed diet, are imperative.

Internal medication may be required, such as calcium salts, ferments or yeasts, ichthyol, salicylates, alkalis, mineral waters or intestinal irrigations. The subcutaneous injection of 0.5 mg. of adrenalin often attenuates the crises and repeated applications have cured a few cases. [Immediate relief is often obtained by the subcutaneous injection of 0.01 gm. of pilocarpin muriate.]

The remarkable efficacy of serotherapeutic treatment in some cases of urticaria has attracted attention to the study of the different forms of this method, but its results are not constant. Sometimes a rapid cure has been obtained by copious venesection followed by intravenous injection of physiological salt solution, by the injection of serum from a healthy person, by autoserotherapy, by the injection of animal serum in very minute doses. The need of caution in the employment of these remedies cannot be overemphasized, as they are liable to aggravate the trouble. The procedures designated to establish an anti-anaphylactic state have not yet been formulated.

External measures, although constantly demanded by the patient for the relief of the pruritus, are of minor importance. Baths and douches, it is well to know, often aggravate the trouble; and similarly ointments. Soothing acid or alcoholic lotions are preferable and should be applied warm or cold, or rather lukewarm, with a decoction of slippery elm, lime, or chamomile, or with a solution of vinegar, lemon-juice, camphorated alcohol, carbolized glycerin, menthol, thymol, resorcin, etc., followed by abundant applications

of neutral powders. The hygiene of the skin requires some attention; the underclothing should be light, of fine smooth texture and must not rub or press on the skin.

Whatever the cause of the urticaria, the treatment should not be schematic but should be specially adapted to the requirements of the case.

GIANT URTICARIA.

Under this name, or that of *acute circumscribed edema* of Quincke, an affection has been described which manifests itself by the sudden appearance of edematous infiltrations, usually fairly well outlined, firm, pinkish or porcelain-white in the center, with a rose-colored periphery. These infiltrations have the size of a hazelnut, walnut, or even an orange; their elevation may amount to several centimeters; they are the seat of a sensation of tension, burning or itching. They may appear at any point of the integument or even of the mucous membranes, although the face and the region of the genital organs are the seats of predilection.

The attack comes on suddenly, often during the night, without prodromata or with some malaise and a slight fever; a single elevation, or a small number, appear and persist for a few hours, at most two days. The attacks may be periodically repeated or they may be separated by intervals of variable length.

This affection persists for years, associated or not with common chronic urticaria; it finally disappears, sometimes to become replaced by other pathological manifestations. Its possible localization in the upper respiratory passages represents the only dangerous element. Also, according to the seat of the edema, the patient may be disfigured for a few hours.

Etiology.—The etiology is the same as that of chronic urticaria. There exist intermediate cases between this and Quincke's edema.

The paroxysms sometimes follow overeating, errors in diet, nervous overstrain or the action of cold. In the interval, the patient may enjoy excellent health.

Treatment.—The treatment follows that of ordinary urticaria. Simple calcium chloride, systematically administered, in courses, has repeatedly proved entirely successful in my experience.

URTICARIA FACTITIA, OR DERMOGRAPHISM.

It has been stated above that in most cases of urticaria, the eruption can be provoked at the time of the attacks by scratching or other cutaneous irritations. Dermographism is an altogether different phenomenon. In certain individuals, a mechanical irritation, especially forcible friction with a blunt point gives rise to

a special cutaneous reflex, a non-pruritic urticarial elevation. A very brief stage of anemia with prominence of the hair follicles, is followed by the appearance of a bright pink line, which widens to 1 to 2 cm., the middle becoming raised in less than a minute in the form of a ridge. At the end of five minutes this ridge may attain a height of 3 to 4 mm. and a breadth of 1 cm.; the phenomenon usually lasts from fifteen to twenty minutes or sometimes several hours. Although electrical stimulation or other irritations may also cause its appearance, a mechanical action is most effective. Friction over the affected region, after the complete subsidence of the phenomenon, may cause the reappearance of the writing or designs which had been traced on the skin.

Fig. 5.—Dermographism. (Ormsby.)

Dermographism is observed especially on the trunk and on the first segments of the limbs. It is rare in the face, but is said to have occurred on the buccal mucosa.

Dermographism is one of the stigmata of the neurotic constitution. It is met with in hysterical individuals, in epilepsy, in 20 per cent. of the insane, especially among idiots and constantly in catatonia; furthermore, in the victims of lead poisoning and chronic alcoholism, especially in consumers of aromatic liquors. In the spring, during the menstrual periods and after emotions or fatigue, dermographism is the most marked. It is noteworthy that it

does not often occur in association with common urticaria. Its name of pseudo-urticaria is derived from the absolute objective identity between the dermographic elevation and the urticarial wheal. The suggestion has been made that the mysterious aspect of this phenomenon may have led to its being used in former days in the tricks of sorcerers. At the present day, malingerers have utilized it for the simulation of some other eruption.

Treatment.—The treatment consists in attention to hygiene, if necessary, and care of the general condition.

CHAPTER III.

PURPURA.

The name *purpura* is applied to an eruption of spontaneous hemorrhagic spots. The spots of purpura are of a bright or bluish red and do not disappear under pressure of the finger; they are usually of rounded shape, flat or slightly elevated, of variable extent, more or less numerous, but always multiple.

Petechiæ is the term in use for the description of small punctiform or lenticular lesions; they sometimes surround the pilo-sebaceous orifices. *Ecchymoses* are more extensive and irregular; they vary in size from that of a coin to that of the hand, or more. A less common term, *vibices*, designates more elongated or striated purpuric spots.

After a few days or weeks, depending on their size, these hemorrhagic spots fade away, after having passed through the same shades, purplish, brownish, greenish and yellowish, as the traumatic ecchymoses.

Petechiæ are highly characteristic, being necessarily spontaneous; ecchymoses may have been produced by a forgotten or wilfully denied *traumatism*, sometimes very insignificant in the victims of hemophilia.

Purpuric spots must not be confused with vascular nevi, which are of indefinite duration; nor with the spots of erythema, which fade away under pressure of the finger.

The combination of erythema with different varieties with purpura, either in the same spot as the hemorrhages or coincidently in the same region, is not unusual. The purpuric spots may also be urticarial at the onset (purpura urticans) or become complicated by urticaria.

We must therefore admit an obvious relationship, transition-forms and various combinations, between purpura, erythema and urticaria (such as *erythema nodosum contusiforme, urticaria hemorrhagica*, etc.).

The term purpura should not be applied to occasional *hemorrhagic eruptions*, such as occur in smallpox, herpes zoster, eczema, pemphigus and the pyodermatitides. Such conditions are properly described as hemorrhagic variola, etc.

The *eruption* of purpura comes on in sudden or prolonged attacks, which are frequently successive. The attack may be preceded by

inflammatory local edema, sometimes lymphangitic, of short duration and accompanied by heat or pruritus, or it may occur without the patient's knowledge. The affected region appears irregularly dotted with lesions, all of the same size or of different sizes, of the same age or at different stages of their development.

The distribution of purpura is often more or less symmetrical; the lower limbs are most commonly and sometimes alone affected, or sometimes all the extremities are involved. The eruption may occupy any location and may even invade the mucous membranes, where it is apt to assume the form of blood blisters which rupture and give rise to hemorrhage.

In some cases of purpura, it is possible to produce a hemorrhagic spot through moderate pressure on the skin with a blunt point (*provoked purpura*), or an eruption of petechiæ through the temporary constriction of a segment of the limb (tourniquet purpura).

Clinical Forms.—The eruption of purpura is sometimes merely a commonplace *symptom* of several pathological conditions; these cases are described as *secondary purpuras*. Again, it may form part of one of the *syndromes* designated as *primary purpuras*, representing their most salient feature.

As a matter of fact, purpura occurs under very different conditions; occasionally in the midst of health and without an appreciable determining cause; or as a sequel of overstrain, or after intoxications; or in the course of definite infectious diseases or cachexias. It may furthermore be associated with a train of general phenomena, among which rheumatoid pains, gastro-intestinal disturbances, fever, general malaise, etc., are especially common.

On the one hand, the purpuric eruptions may occur without hemorrhage from the mucosæ, constituting *purpura simplex*. On the other hand, it may be accompanied by very profuse epistaxis, bleeding from the buccal mucous membranes, especially the gums, metrorrhagia, melena, hematuria or visceral hemorrhages, constituting *purpura hemorrhagica*.

This division, for which we are indebted to Willan, can only be maintained from the descriptive point of view, for it does not correspond to a difference in etiology; a simple purpura may at any time become transformed into purpura hemorrhagica. It is not even in harmony with the prognosis, purpura hemorrhagica being sometimes benign and at other times grave.

It seems more rational to base the establishment of the clinical types on the question of whether the purpura is a simple symptom or whether it constitutes a syndrome.

Great differences of opinion on this point exist among authors. The following forms are generally admitted, although under different designations:

SECONDARY PURPURAS.

The secondary purpuras are those which appear as an ordinary symptom or epiphenomenon in the course of a large number of pathological conditions. They possess an indicative value, but are of little importance in themselves.

Four classes of these purpuras are recognized:

1. *Mechanical purpuras*, which occur on the limbs under the influence of prolonged constriction, or at any point of the body in the course of asystole [in a disturbance of cardiac compensation] or of fits of whooping-cough or attacks of epilepsy.

2. *Toxic purpuras*, which may be provoked by certain medicinal agents such as phosphorus, potassium iodide, mercury, arsenic, antipyrin, chloral, salicylates, quinin, belladonna, ergot, copaïva, etc., as well as by injections of antitoxic sera, bites of venomous serpents, etc.

3. *Secondary purpura of the acute infectious diseases*, such as angina, diphtheria, scarlet fever, gonorrhea, typhoid, typhus, miliary tuberculosis, malaria, etc.

4. *Cachectic purpuras*, observed at advanced stages of serious diseases, cancer, tuberculosis, Bright's disease, hepatic cirrhosis, icterus gravis, pernicious anemia, the leukemias, etc. The eruption usually comes on insidiously, without inflammatory phenomena, sometimes with edema and occupies chiefly the lower extremities.

The group of cachectic purpuras is really most heterogeneous and many cases are probably referable to an infection, auto-intoxication, nervous lesions or anomalies of the blood.

Purpura senilis of Bateman has sometimes been grouped under the same heading. This designation is applied to purpuric spots which occur incessantly, for years, without general disturbances, in aged individuals; located predominantly on the forearms. Unna and Pasini have shown that these cutaneous hemorrhages are related to senile degeneration of the skin.

PRIMARY PURPURAS.

The primary purpuras are those in which the petechiæ and ecchymoses, with or without hemorrhages of the mucous membranes, represent the exclusive or principal phenomenon. Several types are recognized:

1. *Rheumatoid purpura* is the most common; it is identical with Schoenlein's peliosis rheumatica and the myelopathic purpura of Faisans.

It occurs in both sexes but more frequently in youthful or adult males and is supposed to follow on exposure to damp, cold, fatigue,

overstrain or emotional disturbances. The onset is often marked either by a sensation of fatigue or articular pains in the lower limbs, or by a more or less extensive transitory edema in the same regions, or finally by gastro-intestinal disturbances. Fever is variable, not high and may be absent.

The rheumatoid pains are arthralgic, associated or not with articular swelling; they affect the knees and ankles especially, but may extend to the joints of the upper extremities.

Sometimes the pain is muscular or neuralgic. The distinction between these rheumatoid pains and those of acute rheumatism has been emphasized by Besnier.

The *gastro-intestinal disturbances* consist in repeated vomiting with gastralgia, in intestinal colics, often severe, simulating the pains of peritonitis, or accompanied by diarrheal crises, melena, or dysenteriform evacuation. These various manifestations precede or accompany the eruption and are temporary, but may sometimes recur in the course of the disease. Various complications have been noted on the part of the serous membranes and the viscera.

The eruption consists of petechiæ, more or less mixed with small ecchymoses; it affects especially the lower limbs symmetrically (Fig. 6), but may become generalized, beginning with the upper limbs. Not infrequently, it is polymorphous, mixed with papular, nodular, or urticarial erythema. The name of *purpura exanthematica* is used by Laget for this eruptive complex.

The attacks recur at very irregular intervals. They are sometimes so obviously aroused by posture or walking as to justify the expression of "orthostatic purpura." Periodical attacks coinciding with the menses have also been observed.

The majority are cases of purpura simplex. Hemorrhages are rare, but may supervene in the course of evolution, aggravating the clinical picture.

The duration of the disease is ordinarily a few weeks; but some cases are prolonged for several months. Recurrences may take place at irregular intervals.

The severity of the systemic symptoms varies within extreme limits; sometimes they have to be looked for, or, on the contrary, they may have the appearance of a grave infection.

Sporadic scurvy, only rare cases of which are now met with, differs from rheumatoid purpura by its etiology, which involves confinement and deprivation of fresh foods, as well as by the swollen condition of the gums and the well-marked anemia and asthenia. These cases, however, may be interpreted as rheumatoid purpura developing in a prepared soil.

In *infantile scurvy* or *Barlow's disease,* the ecchymosis and hemorrhages of the mucous membranes are far from constituting the most

important feature of the clinical picture, in which painful pseudo-paralysis [due to involvement of the joints] predominates.

Although apparently primary, the cases of chronic purpura, reported especially by Hayem and his school, as well as by Millard and others, are probably of autotoxic origin or connected with a more or less latent infection, such as tuberculosis. The continuous or intermittent cutaneous and hemorrhagic manifestations may be

Fig. 6.—Purpura rheumatoides.

prolonged for ten or twenty years, the prognosis being nevertheless regarded as relatively favorable. This form must not be confused with *hemophilia*, which is congenital and hereditary and in which the coagulability of the blood is always markedly diminished.

Under the name of *purpura annularis telangiectoides*, a well-defined clinical type was described by Majocchi, in 1895, and has since been studied by several authors. It consists of dull red spots, symmetrically distributed on the lower limbs especially, at first telangiectatic,

then hemorrhagic and becoming ring-shaped through gradual centrifugal extension. Pasini attributes these spots to endophlebitis of the small, deep veins of the skin. The etiology of this dermatosis is entirely unknown.

2. *Primary infectious purpuras* are those in which the general symptoms indicate an infection of the type of septicemia. Besides relatively attenuated forms, which form a connecting link with rheumatoid purpura, there occur very grave forms, such as the *angiohematic typhus* of Landouzy and Gomot, and the *purpura fulminans* of Henoch.

Such cases may begin with a severe initial chill, vomiting, a temperature of 40° [104°], typhoid stupor, delirium, coma, dry tongue, albuminuria and sometimes icterus, together with scattered petechiæ and ecchymoses and hemorrhages from various avenues. The purpuric lesions may become gangrenous; cellulitis, purulent arthritis, etc., may occur. Several of these cases have recently been traced to infection by the meningococcus, the presence of which in the blood and in the spots has been demonstrated by A. Netter and Salanier. Death may follow in two or three days in the acute variety; or in one or two weeks, in the typhoid variety.

3. *Abrupt apyretic purpura hemorrhagica,* or *morbus maculosus*—two typical instances of which were published by Werlhoff—is a rare form. According to a generally accepted standard, its behavior is as follows:

In the midst of health, sometimes after an emotion or slight traumatism, often without prodromata or malaise, without fever, a slight nasal or gingival hemorrhage makes its appearance. On the next day, petechiæ are noted on the lower limbs, then larger scattered ecchymoses together with hemorrhages from various mucous membranes. After ten to fifteen days, recovery sets in without complications. Neither infection nor intoxication, nor a grave lesion of the blood have been demonstrated in the course of the disease.

It appears more and more probable that a considerable number of the clinical forms of purpura discussed above, notably the primary purpuras, the secondary purpuras following infectious diseases and even some of the so-called cachectic purpuras, do not constitute distinct diseases, but merely degrees or variations of the same morbid process. Interpreted in this manner purpura, simple or hemorrhagic, would be merely the consequence of various acute or chronic infections, causing alterations in the composition of the blood and vascular or visceral lesions which in turn are responsible for the different aspects of the syndrome.

Pathogenesis.—Purpura is observed in both sexes, at any age, under a great variety of conditions as referred to above.

Pathological Anatomy.—Pathological anatomy teaches that the purpuric spots always contain an *effusion of blood*, with its white corpuscles; situated at a variable level in the meshes of the derma or hypoderm up to the papillæ, where it undergoes the usual changes of extravasated blood. Sack has shown that vascular rupture most frequently occurs in the small veins of the subdermal plexus. The vascular walls, although various changes and degenerations have been reported, are nearly always in a remarkably intact condition.

In the absence of local lesions to account for the hemorrhages, different general conditions have been invoked, which, however, do not exclude one another.

In the circulating *blood*, the number of red cells remains normal, except in case of profuse hemorrhages, or pernicious anemia; the number of white corpuscles is rarely changed (leukemias); the blood platelets—according to Denys, Hayem, Bensaude and others, confirmed by W. Duke—may become very scanty, or even entirely disappear (L. Le Sourd and Pagniez). As to humoral lesions, a retarded or normal coagulation of the blood, a non-contracting or imperfectly contractile and friable clot and an increased red blood cell resistance has been noted. The "bleeding time" (W. Duke) may be greatly increased. However, these various phenomena are inconstant; their importance, mutual relations and pathogenic significance are still under investigation.

Visceral lesions are common. Those of the liver and kidney may be charged with having prevented the destruction or the elimination of certain toxins; on the other hand, the role of the hepatic function in the coagulation of the blood is well known. Changes in the intestinal tract are frequently found in the purpuras, as shown by clinical observation and autopsies; I believe that the intestinal tract is often the starting-point of the infection or intoxication to which the purpura is due, perhaps through intermediation of the liver which is damaged in transit. A form of abdominal purpura has even been described (Henoch). A pathological condition of the suprarenal glands and bloodvessel glands has sometimes been held responsible.

The role of the nervous system is shown by a large number of facts. The rheumatoid pains characteristic of certain primary purpuras have been referred to above. Petechial eruptions have been observed in the course of myelopathies and neuritis. Straus has noted their coincidence with the fulminating or lightning pains of tabes. There are cases of purpura involving only one-half of the body, or even in exceptional cases assuming a metameric or radicular distribution, proving at any rate a localizing influence of the nervous system. Spinal puncture sometimes, but not invariably, reveals a

lymphocytosis of the cerebrospinal fluid. Finally, Grenet has succeeded in the experimental production of purpura in rabbits, by first damaging the liver and then injecting toxins into the spinal cord.

The possible effect of intoxications is illustrated by the existence of the toxic purpuras, which have been mentioned. A probable influence of auto-intoxications and perhaps of microbic toxins may be inferred.

Infection, as I have intimated, is probably responsible for all the non-toxic forms of purpura. Originally asserted by Hayem, this fact has been established by clinical observation and laboratory research in a group of cases and is very probable for the remainder, including the so-called neuropathic, dyscratic, or cachectic purpuras. Moreover, the literature contains a very considerable number of cases where the staphylococcus, streptococcus, pneumococcus, pyocyaneus, meningococcus, etc., have been demonstrated either in the purpuric spots or in the circulating blood. These facts tend to show that a variety of infections may manifest themselves in the clinical picture of purpura.

It is possible, but by no means an established fact, that there exists a specific microbe, more apt than others to produce the pathological picture of infectious purpura. This would serve to explain the cases of epidemic and contagious *purpuric fever*, mentioned by some authors, the occurrence of which, however, is somewhat doubtful.

Treatment.—Any case of purpura, even when its onset is apparently most harmless, must be carefully watched, as it is impossible to foretell when and how it will end. These patients should accordingly be put at rest, under good hygienic conditions, on strict diet and in the open air if possible. For the control of the hemorrhages, the old medication with acids, ratanhia, hamamelis, ergotin, iron perchloride, is now abandoned, experience having shown them to be utterly useless. They have been replaced by modern agents, such as gelatin, calcium chloride, injections of peptones and blood-sera, hepatic and suprarenal extracts, adrenalin, pituitrin, etc.

The results obtained in the treatment of hemophilia and hemoglobinuria have led to experimentation with methods acting on the equilibrium of the blood, modifying its coagulability and producing what Widal has described as the "hemoclasic crisis." Injections of horse-serum, in large amounts, or in moderate or minute repeated doses, as well as autoserotherapy, have their advocates. [A careful search must be made for occult sources of infection with special attention to the teeth and the tonsils.] These methods, which do not exclude adjuvant medication, especially with calcium salts, have yielded very encouraging results, but their indications have by no means been definitely formulated.

CHAPTER IV.

ECZEMA.

The eruption known as eczema is not characterized by a single eruptive element, but by a series of elementary lesions which succeed each other, combine or coexist in neighboring localities. These lesions are the result of an inflammatory process, affecting the epidermis and cutis; an epidermo-dermatitis, comprising several stages which are of equal importance.

Clinically, these stages are manifested under the following aspects: *erythema, vesiculation, exudation, crust formation, lichenization* and *desquamation.*

Histologically, the lesions consist, in the epidermis, of spongiosis, acanthosis and parakeratosis; in the cutis, of congestion, edema and moderately abundant cellular infiltration.

This definition must be supplemented by mention of the three characteristic features of the *eruption:* its usual arrangement in spots, patches, or surfaces with *irregular outlines* (insular, geographical or archipelagous); its development *in crops* or *relays,* with a tendency to *peripheral extension* and often to a chronic state with fresh exacerbations and its more or less *pruritic* character.

Defined in this way, the eczematous process is one of the most easily recognized. It must be appreciated, however, that this process is not limited to a single and specific dermatosis, but on the contrary represents a relatively common mode of *reaction* of the skin toward a series of mechanical, physical, parasitical and microbic irritants. This reaction must be considered in all respects as *inflammatory.*

Eczema, Eczematization, Eczematosis.—The subject of eczema has become extremely complicated and much confusion has been caused by the application of the term eczema for some time to absolutely different conceptions. Eczema was interpreted by Hebra and the Vienna school of dermatologists, as a very common polymorphous affection, which may at any time be artificially produced in any person. According to the French school, on the other hand, eczema was a rare disease, especially in the clinical material of hospitals, incapable of being artificially produced, since it implies the existence of a general predisposition—altered tissue-juices—a diathesis. Hence, two entirely distinct groups of facts had to be dealt with. Besnier, in 1892, was perhaps the first to elucidate

the controversy by creating the word *eczematization* to designate the eczema of Hebra-Kaposi, which is not a disease but an *eczematoid* artificial dermatitis. The eczema of French writers, on the contrary, is a true disease, which although having multiple causes is related to a peculiar condition of the individual; this disease is chronic and recurrent. The eruptive manifestation of eczematous disease is identical with that of eczematous lesions, or eczematization.

The question would thus seem to have become quite clear, but on closer examination considerable difficulties of interpretation are encountered. If several persons be subjected to the same external irritant, for example, rubbing with spirits of turpentine or tincture of arnica, some will experience at the injured point a transitory erythema or a dermatitis in the form of eczematization with a marked tendency to subsidence; in others, this dermatitis will be extensive and may even become generalized, then healing more or less rapidly; and finally, in others, its duration will be prolonged, recurrences supervening on slight causes or even in the absence of an appreciable cause and this abnormal condition may occasionally persist throughout life.

Several suggestions have been made in explanation of these differences in the evolution of the lesions. It has been claimed that an eczematization produced by contact with an irritant, no matter what its evolution and duration, is and remains an artificial eczematoid dermatitis, there being only an objective and apparent identity between it and the disease eczema dependent on a diathesis. This interpretation would necessarily lead to grouping absolutely similar clinical pictures under different headings and to basing the diagnosis on the patient's statements or on an estimate of the irritative properties of the supposedly provoking causes.

It has also been stated that an ordinary eczematoid dermatitis may become transformed into a specific dermatitis; but this can hardly be admitted, as it is not known wherein the specificity of the disease eczema lies.

Obviously, on the contrary, the persons of the first group in the above-mentioned example, were in a state of physiological integrity, investing them with relative immunity toward the injurious agent, whereas, pathological conditions creating a morbid tendency existed in the others. Those who were affected in the most severe and persistent manner were eczematous subjects in whom a local irritation sufficed to bring out a hitherto latent disease. Although this is entirely plausible, it must be added that, keeping in mind the very variable evolution of eczematous dermatitis of artificial origin, one is forced to admit an entire scale of subnormal and abnormal states, creating a more or less marked predisposition for the appearance, persistence and recurrence of the eczematous eruption.

Thus all distinct boundaries or fundamental differences between eczematization or artificial eczematoid dermatitis with a rapid tendency to spontaneous cure and true eczema, a chronic recurrent disease, are obliterated. It is now merely a question of degree and there remains no valid reason for withholding the name of eczema from any eczematization, whether of artificial or of apparently spontaneous origin.

The term *eczema* will therefore be applied in this book to all eruptions corresponding to the definition at the beginning of this chapter. This name will be supplemented by qualifying adjectives, referring to the momentary objective appearance (vesicular, exudative, crusted eczema, etc.), or to the evolution (acute, chronic, recurrent eczema), or to the apparent etiology (artificial, occupational, microbic eczema, etc.).

I propose the term *eczematosis* for the chronic pathological condition described by other authors as constitutional eczema, eczema-disease or true eczema.

I shall speak of *secondary eczematization*, or of an *eczematized* dermatosis, when a dermo-epidermatitis of eczematous type becomes superadded to the lesions of a preëxisting dermatosis (example: eczematized prurigo, eczematized psoriasis, etc.).

Finally, as will be seen in the next chapter, I call *eczematides* the dry eczemas, the eczema seborrhœicum or seborrheids of other authors.

Pathological Anatomy.—Eczema being an inflammatory pathological process clinically manifested by very variable objective appearances, the nature of their process must be studied in detail before describing the manifestations.

1. The chief lesion of eczema is an edema of the Malpighian body; the serous fluid infiltrates between the epidermic cells, stretching their connecting filaments, so that the rete assumes an appearance which is very properly denominated as a *spongioid state*, by Unna, and as *spongiosis*, by Besnier.

2. When in the spongioid state, the fluid has sufficient pressure to rupture the connecting filaments of the Malpighian cells, it collects in *vesicles*, cavities filled with a transparent fibrinous plasma, containing a few wandering cells and bounded by epidermic cells which have been pushed aside and sometimes compressed. These vesicles, which at first are very small, originate in the deep layer of the epidermis; they increase in size and become confluent with neighboring vesicles until they are visible to the naked eye and protrude under the horny layer; ultimately vesicles form at all levels in the rete; the oldest are pushed up toward the surface by the outward growth of the epidermis and new ones are often formed beneath them.

The vesiculation of eczema is therefore interstitial and dependent on the spongiosis, therein differing from the vesiculation of zona, varicella, etc.

The ultimate fate of eczema-vesicles is variable; they terminate by desiccation or rupture, or become secondarily infected.

3. The first contingency, desiccation without oozing or suppuration, may eventuate, no matter what the size of the vesicles, in regions where the epidermis is resistant. It gives rise to tiny crusts or larger crusts, composed in variable proportions of dried serum, the remains of parakeratotic cells and collections of microbes. The epidermis reforms below and the crusts are shed. Desiccation of the foci of spongiosis is the rule in certain forms having a rather peculiar clinical appearance and course, so that they have been separated from the group of moist eczemas under the name of *dry eczemas* or *seborrheal eczemas*. They will be described further on under the name of *eczematides* (p. 90).

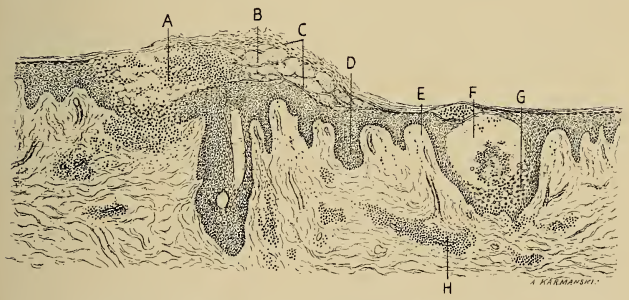


FIG. 7.—Histology of eczema. × 45. *A*, infected vesicle; *B*, crust; *C*, parakeratosis; *D*, acanthosis; *E*, papillary edema; *F*, vesicle; *G*, spongiosis; *H*, perivascular infiltration.

4. When the vesicles have ruptured spontaneously or under the influence of scratching or rubbing, their contents are poured out externally and there is weeping or oozing. In weeping *eczema*, the escape of serous fluid is often prolonged, without production of new vesicles, because the fluid of the spongiosis which is incessantly reproduced, finds a way of escape through the open cavity of the ruptured vesicles; these constitute the *eczematous pores*. The process of keratinization being altered, as will be shown further on, a rapid cicatrization of these orifices cannot occur.

5. The vesicles of eczema may be microbic from the start, or they may be originally sterile. In the latter case, they are easily infected by pyogenic microbes, which find them an excellent culture medium

to which they attract an abundance of polynuclear leukocytes. This is known as *impetiginization* (p. 162).

In these *infected* or *impetiginous eczemas*, the fluid is turbid or purulent and dries in thick melicerous crusts, under which the epidermis remains deeply eroded; moreover, the increased virulence of the microbes, once they begin to grow, leads to a peripheral extension of the lesions and their remote propagation in the form of microbic eczema or of true impetigo.

6. Coincidently with the spongiosis, the Malpighian edema gives rise to the change of keratinization known as *parakeratosis;* this is characterized by the disappearance of the stratum granulosum and preservation of the nuclei in the cells of the horny layer, which contains less fat than in the normal state. In a general way, the parakeratosis controls desquamation and it accordingly predominates in the *squamous, lamellar, psoriatiform,* "craquele," "corné," *hyperkeratotic eczemas,* etc.

7. The spongiosis and the vesiculation, or rather more probably the persistence of the causes which have started and maintained them, lead at the end of a certain time to an abnormal multiplication of the Malpighian cells, hence thickening of the rete; this process is called *acanthosis.* The interpapillary pegs are enlarged and the papillæ are proportionately elongated and narrowed, although frequently edematous. This thickening of the epidermis and of the papillary body results in the appearance of the so-called *lichenoid eczema.* The papillary body in such cases is usually the seat of more or less abundant infiltration.

8. The fluid or plasma of this intra-epidermic edema, which is seen to govern practically the entire eczematous process, is obviously derived from the cutis, where it is exuded from the congested and dilated bloodvessels of the papillary body.

The epidermic lesions described above are accordingly preceded by dermic lesions, consisting of hyperemia and edema of the papillary body, with moderate diapedesis of white corpuscles mixed with a few red cells, which accumulate as a perivascular infiltration. These lesions manifest themselves clinically by redness and swelling, the symptoms being sometimes slightly marked, but in other cases constituting the most obvious phenomenon, for example in acute eczema of the face and in *eczema rubrum.*

Of the various elementary anatomical lesions the most essential are: spongiosis, parakeratosis and acanthosis; all others depend on them. They may become combined or associated, or succeed each other in such a way as to give rise to the multiple aspects which eczema may assume. It must be kept in mind that the qualifying terms vesicular, exudative, squamous, etc., do not designate species

or even varieties of a distinct type, but only accidental, temporary, or more or less persistent phases.

General Etiology.—Considered as a whole, eczema is the most common of all cutaneous affections. Its different varieties and localizations make up nearly one-third of dermatological practice. [In the statistical reports of the American Dermatological Association, covering about three-quarters of a million dermatoses, the eczemas, including eczematides, comprise about 20 per cent. of the total.] It is observed at all ages; during early childhood (*infantile eczema*), then in the period of active life (*occupational eczemas*), finally in the stage of decline of organic resistance (*senile eczema*). The two sexes are equally susceptible and no social class is immune.

Its causes may be divided into external, determining, or occasional and internal or predisposing.

External Causes.—These are innumerable. Nearly all local irritants of any kind, whose moderate influence excites erythema, are capable of causing eczema when their action is augmented. It must be noted, however, that these agents are eczematogenous to a variable degree, some of them requiring a more decided peculiarity or coöperation of the organic soil. Predisposition therefore has a bearing not only upon the extent and duration of the reaction, but upon its character also.

Among the local factors of eczema must be mentioned mechanical agents, such as scratching; physical agents, such as a bright light (Wilson's *eczema solare*), heat (*eczema caloricum*); and countless chemical irritants (arnica, turpentine, phenol, etc.). The majority of these injurious factors will be considered in the chapter on the artificial dermatoses (XXIII). Only a few causes of particular interest will be discussed in what follows:

Traumatic Eczema from Scratching.—Eczema causes itching; the scratching of the eruption, often evidently aggravates it and in some cases may be responsible for its extension and dissemination. The question arises under these circumstances whether the scratching served to transport the irritative agent or the microbes which had invaded the primary focus, or if the skin was directly eczematogenous as a result of individual prediposition. This dissemination was regarded by Kaposi as the effect of an eruptive reflex. In another group of cases, *scratching is primary* as compared to the eruption; this is the case, for example, in pruritus and prurigo (pp. 481 and 488), in scabies and pediculosis, also I believe in ichthyosis, in the circulatory disturbances of the lower limbs, etc. There exists accordingly a true traumatic eczema.

Parasitic Eczemas.—Aside from pruritus, or under conditions where scratching is impossible, parasitic affections may give rise to

genuine eczematization. The eczemas of scabies and pediculosis may therefore be referable either to scratching or to the virus of the parasites.

In trichophytosis of hairless parts, the type known as *herpes circinatus* where pruritus may be almost entirely absent, vesicles due to spongiosis are observed, followed by parakeratotic desquamation. It seems to me justifiable to consider these as eczematous and to attribute them directly to the fungus in the majority of the cases.

Microbic Eczemas. — The question as to the part played by microbes in eczema has been raised only relatively recently. Twenty-five years ago nobody suspected that it would ever arise; and certain modern writers have probably exaggerated its importance.

Unna regards eczema as a *microbic dermatosis;* the non-microbic affections which simulate it are eczematiform eruptions, for the most part artificial dermatitides, but not true eczema. The pathogenic parasite of eczema in his opinion is the *morococcus,* or the morococcic group; this is the name applied by him to the cocci which are found in mulberry-like collections under the roof of the vesicles. Later studies have identified these morococci with certain staphylococci (p. 536).

As a matter of fact, it has been established by careful investigations—among which those of Veillon, Sabouraud, Hallé and Civatte are specially mentioned because I personally assisted in their work—that the vesicles of dermatoses which must be regarded in all respects as eczemas, are *primarily amicrobic,* in the case of artificial eczemas as well as of constitutional eczemas. These vesicles may become infected secondarily by the ordinary microbes of the skin.

On the other hand, these microbes are undoubtedly capable of exciting the reaction of eczematization when they invade the epidermis through the agency of mechanical, physical or chemical traumatization, or of macerating lesions of the protective horny layers; or of an abnormal structure of this layer and its adnexa, as occurs in ichthyosis and especially in kerosis (XI); or of local or regional circulatory disturbances, as in varicose eczema; and finally, even on the normal skin, when they have acquired an increased virulence through culture, in impetigo or any kind of wound, fissure, fistula, etc.

In *eczema* of *war wounds,* described by me as *paratraumatic eczema* (p. 73), countless instances of which have recently been met with, these various conditions were generally associated; the lesion of the epidermis produced by iodin, peroxide water or other antiseptics, maceration through the dressings, local circulatory dis-

turbances, contamination of the surface by the pus of the wound or fistula, combining to produce a genuine type of microbic eczema, more or less impetiginous.

The facts adduced in favor of the general parasitic theory of eczema (Unna, Leredde) are capable of other interpretations. This is true for the auto-inoculation of eczema by scratching, etc.; its recrudescence from imperfectly extinct foci; the peripheral extension of eczematous patches; the identity of eczematous processes irrespective of the apparent causative agent; and the sometimes easy curability by germicidal local applications. But these facts nevertheless constitute a set of impressive arguments, to which may be added a few observations, due to Unna, of positive inoculation of microbic cultures derived from eczema, which produced an eczematiform lesion in healthy inoculated subjects.

Briefly, there exists an assembly of clinical data and laboratory findings, which prove the existence of an *eczema of microbic origin* and character. It is certain that various microbic species, especially belonging to the staphylococcic group, may be eczematogenous. Perhaps there exists a still unknown microbe which more than others possesses this property. At the present writing, however, it is not possible to state that definite species of microbes correspond to the different forms and types of eczema.

We are especially ignorant of the degree to which infection is responsible for eczema (for example in the artificial dermatitides, in eczema from scratching, in parasitic eczema, in the dissemination of eczema), for up to the present time the clinician can no more recognize in a given case whether the eczema is microbic or not, than the bacteriologist can determine whether the detected microbe is primary or of secondary implantation.

Internal Causes.—The general pathological conditions usually met with in cases of eczema, and which may be suspected of being internal or predisposing causes of this dermatosis, are the following: Heredity is often invoked and is said to be either direct, when the patient's parents were likewise eczematous, or indirect, when they suffered from obesity, diabetes, gout, rheumatism, lithiasis, asthma or migraine—briefly, from one or more of the multiple manifestations of the nutritional disturbance known [in France] as *arthritism*. In other cases, the parents were inebriates, or exposed to various intoxications, neurotic, overworked, etc.

The hygienic antecedents of the patient himself are sometimes distinctly bad, whether in regard to the abuse of stimulants, alcohol, coffee, tea, tobacco, etc., or to an excessive nitrogenous diet, overeating, or food of an inferior quality, or to nervous strain in all its forms.

Still more frequently, auto-intoxications are responsible, due to

gastric or intestinal dyspepsia, chronic enteritis, constipation, etc. I have often traced the trouble to a bad condition of the teeth, which at the same time causes intoxication by the putrefactive products of dental caries and pyorrhea alveolaris, and favors abnormal gastro-intestinal fermentation through incomplete mastication and insalivation of the food. Sometimes, an insufficiency of the emunctories is properly held responsible, such as renal, hepatic or intestinal insufficiency, or a deficient function of the organs of internal secretion. Whatever may be the pathogenic mechanism, the relation between the dermatosis and these functional anomalies is entirely admisssible. Urinary examinations unfortunately yield no concordant results, rendering futile the hope of a coming "urinary formula of eczema."

Nutritional disturbances, the hereditary influence of which has been referred to, are naturally still more injurious when affecting the patient himself. Sometimes, as the patient is sure to emphasize, an indisputable alternation or substitution is noted between the attacks of eczema and certain symptoms such as asthma, neuralgias, migraines, bronchial catarrhs, or digestive disturbances. An obstinate progressive eczema frequently precedes by several months or years, and so to say intimates, the manifestation of a latent visceral cancer. Diabetic patients are evidently predisposed to eczema which not uncommonly begins in the genital regions.

The part played by nervous disturbances is equally apparent. A predisposition to eczema is created, not by central or peripheral nervous lesions, but rather by neurasthenia, overstrain, emotions, moral shock, grief, etc. We do not know if the nervous system acts through a vasomotor or trophic disturbance, which would already be the first stage of the eczematous process; or if it acts merely by lowering the resistance of the skin. At any rate, it seems to be a loss of nervous balance which brings about the excessive irritability of the eczematous areas which show themselves intolerant toward any active form of medication.

A reflex nervous action on the skin has been held responsible for the temporary predisposition to attacks of eczema caused by the eruption of teeth, by menstruation, pregnancy, lactation, the menopause, etc.

Regional circulatory disturbances, like those accompanying varicose veins, for example, may create a manifest local predisposition to eczema.

The **pathogenic mechanism** according to which these manifold causes act is necessarily extremely variable. To formulate it in a few words, one would be tempted to say that as the process is inflammatory; it represents a defense reaction of the organism against the injurious agents which attack the skin.

Although barely admissible for the external causes this general idea would not cover the internal causes. Too many decisive arguments refute the theory, which is still occasionally admitted, however, according to which the skin acts vicariously for other emunctories when these are insufficient, and becomes eczematous under the influence of autogenous poisons, toxins and waste products, which seek an outlet through the skin.

A rather attractive theory was advocated by Jacquet, who claimed the intervention of a nervous reflex action of trophic character. Visceral lesions or internal disturbances are supposed to modify the nervous impulses transmitted to the skin, the resulting trophic change enabling the skin to react in the form of eczema against strong or even very weak external irritants which would have been readily tolerated in the normal state.

The majority of eczemas undoubtedly result from a combination, in variable proportions, of external and internal factors. It is therefore imperative, in a given case, to ascertain through careful clinical analysis the "etiological dominant" and the accessory causes. Only when this requirement is complied with can a rational treatment be instituted with some prospect of a successful outcome and the prevention of recurrences.

Symptoms.—When eczema appears suddenly, on a rather extensive surface, or during an exacerbation in the course of chronic eczema, certain general symptoms may be noted, such as digestive disturbances and especially malaise, excitement, insomnia, prostration and a slight fever.

The *eruption* is essentially polymorphous; it consists of redness, vesiculation, exudation, crusts and scales. Its most characteristic feature has very properly been stated to be vesiculation, but certain eczemas run their course without at any time presenting demonstrable vesicles.

In a general way, but remembering that, as a rule, several stages exist together, the succession of the phenomena may be described as follows: At the onset, vivid reddening with diffuse margins makes its appearance, showing a very finely granular surface on inspection with a lens, with more or less edema, tension and pruritus; representing the erythematous stage. It is rare for the redness to be absent or to escape observation and for the vesicles to appear on healthy skin. The edematous redness sometimes, after persisting for a few hours or a day, fades away and leaves behind a fine lamellar or furfuraceous desquamation, this being observed especially in the face and the genital regions.

As a rule, a few hours after the onset of the attack, a large crop of very superficial vesicles makes its appearance on the erythematous surface, with transparent contents, from the size of a needle-

point to that of a pin-head, very close together, sometimes becoming confluent in bullæ of moderate size; this is the vesicular stage. On the hands and feet, in regions where the epidermis is thick, the vesicles are deeper and have less tendency to rupture. They may dry in crusts which are gradually eliminated.

Usually the vesicles do not exist long without rupturing spontaneously or under the influence of scratching and permit the escape of a clear, slightly stringy fluid, faintly yellowish or turbid, stiffening the underwear like mucilage. The exudative stage may be prolonged for several days or even weeks in different cases

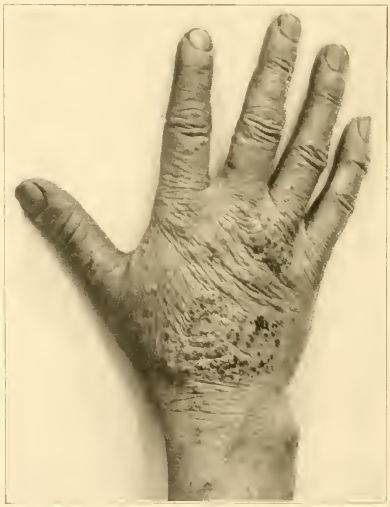


Fig. 8.—Eczema vulgare with vesicles and crusts, showing eczema "pores."

When dressings are employed, the surface of the skin, on removing the dressings, is seen to be red or a vivid pink, smooth and perforated with superficial round or polycyclic erosions from which droplets of a clear sticky serous fluid exude. In case no vesicles have been demonstrable, these erosions which are the eczematous pores previously referred to, represent their equivalent (Fig. 8).

In the absence of dressings and when the exudate is not very profuse it dries in thin yellowish crusts, or in brown concretions when a little blood is mixed with the serum; constituting the stage of incrustation, or *crusted eczema*.

When a pyococcic infection becomes implanted on the eczematous surface, which is not uncommon in children or in certain regions of the body, the secretion is mixed with pus, the crusts are melicerous or grayish, thicker and rougher. The surrounding areas present the features of genuine impetigo and the case becomes one of *impetiginous eczema* (p. 82).

The exudate lessens and dries up after a variable time; the crusts fall, the surface becomes covered with epidermis; but the new horny layer remains thin, transparent, slightly adherent; it cracks through desiccation (*fissured eczema*), and is shed in lamellar or furfuraceous scales which are incessantly renewed.

This stage of desquamation may last a very long time. The vesicles are very apt to reappear on the pinkish scaly surface, separately or in groups, continuously or in crops, with a renewal of the the exudation and crust formation.

The persistence of the eczematous process, aggravated by scratching, the local conditions of the affected region and the general health of the patient, tends to produce a thickening of the patches, with elevation and induration. The normal folds and lines of the skin are more pronounced, the surface is dry and roughened, scaly or crusted. This condition is designated as *lichenified eczema.*

Eruptive Varieties.—It is uncommon for the various stages of the eczematous process to follow a regular course; as a rule, they intermingle, blend and exist together in the same patient. However, it must be admitted that certain eczemas, or rather the eczemas of certain patients, preferably assume one or other objective form and persist a long time at one of the stages, the characteristics of which are then especially well marked.

The varieties of the eruption which require special mention are the following:

Vesicular eczema, with constantly renewed vesicles, occurs especially on the extremities. A special type has been distinguished under the name of dysidrosis.

Exudative eczema, with continuous exudation, is observed in gouty or obese persons especially on the legs and arms, in the newborn on the face, sometimes as the result of irritative applications.

Eczema rubrum is the form in which the reddening is intense (often edematous) and does not completely disappear under pressure of the finger, indicating an abundant diapedesis of red corpuscles in the cutis. It occurs in extensive patches in acute attacks, or is persistent and indolent, on the legs, on the large folds and also in the face.

The term *erysipeloid eczema* is sometimes employed to designate sudden inflammatory and edematous attacks, sometimes followed by vesico-bullæ, affecting especially the face and the genital

organs. It is frequently a complication of a dry sluggish eczema, or the result of an artificial local irritation.

Dry eczema, which in exceptional cases is arranged in circumscribed spots or patches, often polycyclic, with a pinkish scaly surface, without demonstrable vesicles, is so common and so peculiar in its appearance and behavior that there is a strong temptation to place it in a group by itself (seborrhœic eczema, seborrheid, psoriatiform parakeratosis, etc.). It will be discussed at length under the name *eczematides* in the chapter on the erythemato-squamous dermatosis (V, p. 90).

Squamous eczema, in which desquamation is abundant and continuous, is encountered especially in individuals or regions with defective nutrition.

Horny, or *keratotic* or *tylotic eczema* is practically limited to the plantar and palmar regions (XI, p. 214).

Lichenified eczema, often circumscribed, very pruriginous and chronic, seems to be likewise connected with special local conditions. Its differentiation from eczematized prurigo is sometimes very difficult (p. 489).

Impetiginous eczema, more common in children and in the artificial dermatitides may give rise to folliculitis, furuncles, adenitis, lymphangitis and abscesses, in short, to all the manifestations of pyodermatitis (p. 82).

Varieties of Configuration.—The distribution and extent of eczematous eruptions are extremely variable and not easily classified. The most usual configuration is in form of spots, patches or surface lesions of very unequal size and quite irregular contours, in geographical or archipelagous designs. This type, designated as amorphous eczema by Devergie, is entitled to the name of *eczema vulgare.*

The so-called *papulo-vesicular eczema* of Brocq is characterized by the fact that its initial element is a small papulo-vesicular elevation, instead of a simple vesicle; the confluence of these lesions results in patches with an indurated base. The eruption is generally widely scattered and develops in successive crops. There is intense itching and the relation of this form to the prurigos is very evident in certain cases. Frequently, however, this variety is combined with eczema vulgare in other regions of the body.

Nummular eczema assumes the form of round or oval, sharply circumscribed spots, which are sometimes herpetoid (Unna) or trichophytoid (Sabouraud). It is common on the wrists, on the dorsal aspect of the hands and on the legs. [The patches are remarkable for the absence of peripheral extension, their recurrence throughout a number of years and the association of moderate eosinophilia.]

Varicose eczema and *paratraumatic eczema of war wounds* are noteworthy on account of their distinctly outlined borders, their

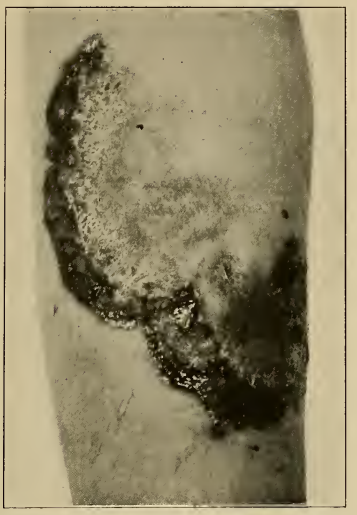


Fig. 9.—Paratraumatic eczema, following on a bullet-wound of the right arm of three months' standing, cured in fifteen days. Noteworthy case on account of the arciform and syphiloid configuration of the eruption. Note the central cicatrization and the extension by vesico-pustules, especially on the convexity of the border at the base of the figure.

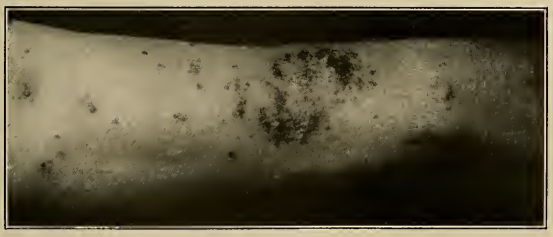


Fig. 10.—Eczema of the papulo-vesicular type on the forearm.

usually polycyclic configuration, and their continuous peripheral extension; they are apt to be combined with impetigo.

The *eczema marginatum* of Hebra is a parasitic epidermatitis due to the epidermophyton (p. 524).

Disseminated vesicular eczema will be considered in a paragraph by itself (p. 87).

Generalized eczema will be discussed with the erythrodermas (p. 120) and *eczema follicularum* in the chapter on the folliculoses (p. 396).

FIG. 11. Recurrent eczema of the face in a girl aged six years.

Regional Varieties. On the *scalp*, especially in children, eczema is often incited by pediculi and associated with pyodermatitis; in youthful individuals and in adults it is ordinarily a complication of pityriasis and there are imperceptible transitions between dry pityriasis, steatoid pityriasis, dry, exudative and crusted eczema (see Kerosis, p. 196).

In the *beard* and *hairy regions* the same relations with pityriasis are noted. The eczema may be of the exudative type and often gives rise to pyococcic sycosis.

Orbicular or *orificial eczemas* are dry or exudative, often very obstinate and are caused or maintained by lesions of the mucous membranes and corresponding cavities; they have been interpreted as of reflex origin. On the lips, a bad condition of the teeth, irritating tooth-pastes, pharyngitis, etc., may be responsible.

Persistent desquamation of the red border of the lips forms a special type of unknown character which has been referred by some writers to psoriasis, seborrhea, etc.

On the nostrils, eye-lids and ears conditions are involved such as chronic coryza, sinusitis, ocular disturbances and otitis media or externa; in eczema of the vulva, the trouble may be due to diabetes, cystitis, vaginitis, or metritis; in eczema of the anus, to hemorrhoids, fissures or constipation.

Perigenital and peri-anal eczema very frequently follow on pruritus of these regions.

Generalized eczema of the face, extending to the neck and thorax, is often incited in adults by dyes for the hair and beard, or is the result of an occupational dermatitis. In children it is often recurrent and related to digestive disturbances or the eruption of teeth.

Eczema affecting the *nipple* and *areola*, in women, is often weeping, limited, very obstinate and almost exclusively produced by scabies (Fig. 150), or pregnancy and lactation. It must be carefully differentiated from Paget's disease of the nipple.

Eczema of the large folds localized in the articular, submammary, subabdominal and intergluteal folds, etc., is not uncommon in obese, diabetic or gouty subjects; it is exudative and diffuse, or dry and marginate.

Intertrigo eczema of infants (p. 31) represents a higher degree of irritative reaction than simple intertrigo erythema.

On the *legs*, eczema is observed very frequently and in all its forms; it is associated with varicose veins, traumatic lesions, ulcers, etc., and leads secondarily to dermatoscleroses and hypertrophies.

On the *hands*, *wrists* and *forearms* the majority of eczemas are caused by external occupational toxidermias; further on I shall discuss dysidrosis, which affects also the feet.

Between the toes, it is common to find a red pruritic dermatitis, with exfoliation of large macerated horny shreds. This form has been grouped under eczema, dysidrosis or intertrigo by different writers; Sabouraud has shown that it is usually parasitic and due to the epidermophyton inguinale.

The question of eczema of the mucous membranes is a mooted one. Undoubtedly, eczema exists on the semimucosæ, the red border of the lips, glans penis, labia majora and minora. But in the mouth, on the tongue, in the nasal fossæ, on the conjunctivæ and in the vagina, the reactions caused by eczematogenic factors

are clinically and histologically different from those noted in the skin.

Course and Prognosis.—The course of eczema in general is extremely variable. Authors recognize an *acute* and a *chronic eczema*, according as the eruption appears abruptly and subsides in a few weeks, or becomes locally established and recurs during a period of months or years. This distinction is arbitrary and useless.

The prognosis of a given eczema depends in part on its cause and in part on the patient's state of health, namely the degree of predisposition present; the latter is often recognized only *a posteriori*.

In a general way, it may be stated that it is the nature of eczema to advance in attacks or relays (ἔκζεμα, ἐκζειν, to boil over or effervesce). Eczema begins most frequently but not always with a sudden hyperemic attack with general malaise in its course; it is the rule that new paroxysms and rapid extensions occur at variable intervals, readily provoked by external or internal disturbances. This "tendency to react toward irritants by an increased exudation and inflammation" is so characteristic that Unna included it in his definition of eczema.

During the intervals of the attacks there may be progressive improvement, or simple persistence, or progressive aggravation; or pyodermic, lymphangitic, etc., complications may make their appearance.

Eczema may constitute a real infirmity, preventing employment in a number of occupations. The pruritus and desparation caused by it have sometimes resulted in cachexia or even led to suicide. Fatal cases for which eczema has been held responsible, are probably referable to visceral diseases of which the eruption was merely one manifestation (see Eczematosis).

Diagnosis.—It would be an endless task to point out all possible errors. Usually, moreover, the diagnosis of eczema is clear, through the demonstration of a polymorphous epidermodermatitis, assuming one or several of the above described clinical aspects and arranged in spots or patches with irregular margins. Vesicles should not be credited with a greater diagnostic value than the other elementary constituents, the process *as a whole* being the characteristic feature of the disease.

Greater difficulties arise in elucidating the *cause* and *origin* of the eczema. The points to be determined are these: Is the eczema purely external and, as it were, traumatic; is it primary, originating in the healthy skin, or secondary to a preceding dermatosis; what is the part played by predisposition, local or visceral affections or the general condition of the patient? The beginner's task will be facilitated to some extent, I hope, by the comments which follow on some of the principal types.

Treatment.—Limitation is imperative in a field of such magnitude, so that only a few brief practical directions will be presented.

All cases of eczema should be treated because the patient is uncomfortable and is threatened with the danger of the eruptions, extending as well as becoming infected. The dread of internal "repercussions" is largely imaginary; although experience has shown that in very extensive eczemas, in those due to grave organic or nutritional changes, or which show evidence of vicarious or substitution phenomena mentioned above, it is advisable to apply only clean dressings or very mild soothing lotions and to avoid all active medication.

Two important rules are as follows: In every case of eczema, the physician must in the first place *analyze the probable causes* of the eruption, systematically and with strict attention to detail. To begin with, external irritants are looked for and eliminated as far as possible; next, preëxisting dermatoses, with their marked bearing on the prognosis and treatment and finally, internal causes, which should be controlled by means of appropriate measures.

The second, equally important rule is that the treatment of eczema must be symptomatic and flexible, rather than systematic and predetermined. By this statement is meant that the eruption should be treated according to its behavior, instead of blindly applying one or other formula or medication of "anti-eczematous" repute.

Local Treatment.—Acute eczematous attacks require complete rest, or at least rest of the affected region, with a prescription of simple lotions or spraying with vegetable infusions, or very mild antiseptic solutions and generous applications of neutral powders which favor desiccation; these can be alternated with cooling creams. Salves and pastes are all objectionable at the beginning of an acute attack, as they have a tendency to "heat" the skin and to macerate the epidermis.

In the presence of a crusted or impetiginous eczema, the crusts should always be removed in the first place and this is accomplished by means of lotions with a vegetable or astringent decoction, such as very dilute Alibour water (see Therapeutic Notes § 2), or the application of dusting powders, preceded if necessary by moist dressings [with Liq. aluminis acetatis, 1 : water 10] or aseptic softening poultices. When the surface is clean and the inflammation has subsided, treatment is begun, while continuing the use of lotions twice daily—with applications of ichthyol pastes, with a small addition of resorcin or yellow oxide of mercury pastes; sometimes bland powders, glycerol of starch or watery pastes prove useful.

In cases of oozing and itching eczema, painting with an aqueous solution of silver nitrate (1 : 10 to 1 : 30) repeated every two or three days, is sometimes effective.

When the eczema is only slightly inflammatory, without exudation, but squamous or lichenoid, treatment may begin—after thorough cleaning with vaseline or as is my custom, with petroleum ether—with the series of pastes, then ointments, made with tar, sulphur, weak or strong reducing agents, mercurials, etc., starting with the weakest and passing up to pure tar, topical applications of chrysarobin or pyrogallol and compound plasters. In case an excessive reaction follows, milder measures should be temporarily resumed. The most experienced dermatologists usually employ strong topical applications very guardedly. The recommendation to make use of only the simplest and best tried remedies cannot be overemphasized.

The treatment of dry, psoriatiform and hyperkeratotic eczemas, and of pruriginous eczemas, is the same as that of other dermatoses presenting the same characters.

Mention must finally be made of a few topical applications of more exceptional employment, which sometimes yield very favorable results.

Continuous and direct applications of caoutchouc, or better of caoutchouc-covered cloths, have enjoyed a considerable vogue, but they are often badly tolerated and it is difficult to determine just when their employment should cease.

Contrary to what might be expected strong compound ointments, such as those [of tar and sulphur or even tar and chrysarobin] of the type of Baissade's balsam (Therapeutic Notes, § 7) [or Dreuw's ointment] are efficient not only in the obstinate forms, but also in the case of acute and weeping eczema. Dind (of Lausanne) has shown that crude washed coal tar can be employed under the same conditions; painted on, then dusted with talcum powder, it forms on drying a coating which is left in place for three to eight days; this inexpensive, convenient and highly keratoplastic agent has been generally adopted and has rendered excellent service.

The indications for electrotherapy in the form of static baths or high-frequency currents are imperfectly understood. These procedures are less valuable than radiotherapy, which possesses a high degree of efficiency in very pruriginous and lichenoid eczemas as well as in the eczematized prurigos. However, it is resorted to only exceptionally in localized and very obstinate cases. [The *x*-rays are valuable in all forms of eczema except the acute, not only for their antipruritic effect but also for their action on the infiltrations.]

General Medication.—There is no specific for eczema, but in nearly all these patients, it is necessary to intervene for the regulation of hygienic conditions and the control of the existing tendency to nutritional or visceral disturbances, if any such be disclosed.

The diet, restricted in quality as well as quantity, should be that of the congestive and pruriginous dermatoses of autotoxic origin. A vegetable or milk diet is sometimes necessary.

Constipation and digestive disturbances must be carefully regulated. It is sometimes essential to train the patients to eat slowly and chew their food well (bradyphagia); it may be necessary to insist on the services of a competent dentist for the repair of teeth or the fitting of dental plates.

General hygiene, a quiet life, physical, mental and moral rest and sojourn in the open air, if possible, are of course very desirable.

As to internal medicinal treatment, this varies in different cases and may consist in the prescription of alkalis, cod-liver oil, calcium salts, phosphates or phosphoric acid, iron or arsenic; exceptionally, in opotherapeutic remedies (such as thyroid, suprarenal, hepatic, ovarian, intestinal extracts, etc.).

Arsenic medication, formerly considered indispensable in every case of eczema, has greatly fallen in repute and as a matter of fact, arsenic is more apt to be injurious than useful, especially in the acute forms. As a tonic it may be advantageously employed in sluggish eczemas and in weakened nervous patients. I agree with the majority of dermatologists, in preferring the sodium arsenite to the cacodylates, methyl arsenites or analogous preparations.

It is difficult to state in a few words what mineral waters are suitable for eczematous patients. In a general way, the lymphatic group may be referred to sulphur springs; the gouty to alkaline and silicate springs; nervous irritable persons are benefited by waters charged with calcium sulphates; nervous patients suffering from auto-intoxication do well on so-called neutral and sedative waters; long-standing cases of the dry and lichenoid type may employ arsenical waters.

Some of the most frequent *clinical types* of eczema are entitled to a special discussion:

ARTIFICIAL ECZEMAS.

Their special features depend upon their cause, localization and evolution.

The irritants most apt to incite an eczematous reaction and the occupations which most often lead to its occurrence are discussed in the chapter on the artificial dermatitides (pp. 463, 467).

The artificial eczemas naturally develop first of all in the regions directly exposed; on the hands, especially in the interdigital folds, on the wrists and forearms, in cases of occupational eczema (see Figs. 8 and 144); on the face and neck, in dermatitis due to dyes, etc.

They have a tendency to spread by peripheral extension, but may also advance in leaps, reaching, for instance, the face, the neck, the thighs or the genital regions.

Several explanations of these transferences have been suggested. The injurious substance was assumed to have been transported, for example, by the patient's fingers, which is possible in certain cases; or the toxic agent was believed to have undergone absorption, manifesting its effect from within outward, in predisposed regions; again, the eruption at a distance was interpreted as the result of a reflex action, although it is hardly likely for a true inflammation to be the result of a purely nervous pathogenesis. The most plausible explanation, in my opinion, is that which regards the [reflex] diffusion of the pruritus as the predisposing cause and the scratching or the transportation of the microbic invaders of the first eczematous focus, as the determining cause.

The artificial eczemas are often distinctly vesicular from the start; or they may be erythemato-edematous or even erysipelatoid, quite at the beginning, becoming vesicular secondarily. They are very commonly infected by pyococci and become impetiginous in places, while elsewhere they become keratotic, present fissures and cracks, become covered with crusts and assume a nummular or lichenoid appearance. Extraordinarily polymorphous but nevertheless characteristic appearances are the result, such as those to which special names like grocers' itch, bricklayers' and cement-workers' itch, etc., have been given.

Sometimes, sublata causa, a cure follows with the greatest rapidity and almost without treatment; cases of this kind are described as *tranmatic eczematiform dermatitides*. However, all intermediary degrees occur between this contingency and that of prolonged persistence, with recrudescence without apparent cause and with invasion of and establishment in remote areas, representing a true eczema originating from a focus of local irritation.

Even when a more or less rapid cure is obtained, the patient remains predisposed to recurrences under the influence of the same cause or analogous causes through the effect of a sensitization which has been compared with anaphylaxis; and a change of occupation may become imperative.

INFANTILE ECZEMAS.

In little children, especially between the second to the eighth month, eczemas of rather peculiar etiology, topography and evolution are observed with great frequency (in 5 or 10 per cent. of children in Paris). These children are apt to have eczematous, neuropathic, intemperate or overworked parents. Their diet,

whether they are breast-fed or raised by hand, in the presence or absence of evident digestive disturbances, may be injurious by its quality, its too frequent repetition and especially, as has been shown by Marfan, through an oversupply of food. It must be watched and regulated according to the requirements in a given case. Weaning, when carefully managed, is not particularly dangerous in infantile eczemas and may on the contrary lead to a cure. The part played by the eruption of the teeth, although it has been exaggerated, is evident in many cases; it acts by the congestions, nervous condition and digestive disturbances with which it is associated. Various local causes, bad hygiene of the skin, an excessive use of soap, coryza, impetigo, vaccination, etc., may all serve as determining factors.

Infantile eczema affects preferably the face, especially the cheeks, the forehead and the lips, while the nose and chin often escape. Sometimes it begins on the healthy skin in a distinctly vesicular form, followed by oozing; in other cases it originates in the sebaceous ducts of the scalp and forehead, where it is known as "milk-crust," forming a continuous series from pityriasis simplex to dry, impetiginous, or even exudative and crusted eczema; sometimes again, although the two preceding forms are likewise, as a rule, accompanied by erosions, the eruption is obviously secondary to a primary localized pruritus and to the scratching provoked by it.

This eczema may remain localized, or it may extend as far as the buttocks, the limbs and the trunk. The general condition often remains excellent and the eruption usually stops and disappears about the middle or end of the second year.

Its etiology, localization and course differentiate true infantile eczema from eczematiform dermatitis, usually starting from the buttocks, which has been referred to above; the latter represents merely an advanced degree of intertrigo-erythema and is entitled to the designation of intertrigo-eczema.

SECONDARY ECZEMATIZATION.

Eczematization which develops on the soil of other dermatoses and is provoked by the same, seems to be the cause of much confusion in the classical picture of eczema. Familiarity with this mode of origin of eczema which depends on the so-called pre-eczematous dermatoses is of the greatest practical importance for the clinician as in a large number of cases it enables the clinician to make a correct diagnosis and prognosis and to institute preventive treatment. Moreover, on the assumption that eczema is merely a mode of cutaneous reaction, the theoretical explanation of these facts meets with no insurmountable difficulties.

Eczema and Impetigo.—The relations of impetigo to eczema are complex and have given rise to various interpretations. Clinical findings are rendered intelligible on the basis of the following considerations:

Impetigo or superficial pyococcic infection of the epidermis is very frequently grafted on an existing eczema, whatever its origin, external and artificial or internal and constitutional. The result is a modification of the clinical aspect of the eczema, the secretion becoming purulent and the crusts melicerous and coarse, constituting impetiginized eczema. The neighboring lesions or autoinoculations, the result of scratching, may be the lesions of eczema or the pustules of impetigo, or a mixture of the two.

Conversely, impetigo may give rise to eczema, in this sense that the pyococcic agents of impetigo under certain conditions (probably when they possess a high degree of virulence) and in certain regions, are capable of giving rise to epidermodermatitis of the eczematous type like other irritants. This microbic eczema, which might be called impetiginous eczema, unlike impetiginized eczema, lacks constant characteristics and I believe it may assume the aspect of a vesicular or oozing, crusted or squamous eczema, or even that of dry eczema. The investigations of Sabouraud have even led to the conclusion that pityriasis simplex may be regarded as a "dry impetigo."

This second proposition will hardly be admitted offhand; although it is based upon a number of histological and bacteriological examinations carried out in my laboratory with careful analysis of clinical cases and is, I believe, in harmony with the existing facts.

Eczema and Kerosis.—The pathological conditions of the epidermis known as *pityriasis* and *seborrhea*, grouped by me under the heading *kerosis* (p. 196), create a territory peculiarly predisposed to eczematization. Most commonly, an eczematide or dry eczema develops under these conditions, but this dry eczema has a marked tendency to react in the form of exudation and extensive inflammation under the influence of scratching, injudicious treatment, or other local and general causes; constituting the eczematized seborrheid of some authors.

Vesicular, oozing, crusted, impetiginous eczema may likewise become established from the start in cases of kerosis. The sites of predilection of kerosis, primarily the hairy scalp, the face and the large articular folds, are naturally also the usual starting-points of these eczemas.

The kerotic origin of eczema is so common that according to Unna, the great majority of eczemas would be suppressed by treating and curing seborrheal eczema in its early stages.

Eczema and Prurigo.—Pruritus and prurigo, diffuse or localized, of variable origin, are conditions very apt to become eczematized and the starting-point of more or less extensive and generalized eczemas. Scratching, through its mechanical action and the resulting microbic inoculation, constitutes the pathogenic factor in this complication, which is met with in the urticarias and in scabies. However, not all scratched prurigos become eczematized, but have rather a tendency to lichenize, indicating that a certain degree of local or general predisposition is necessary.

It must not be overlooked, on the other hand, that primary eczema is itself pruriginous and becomes lichenized under certain conditions of persistence, localization and soil. The relations of eczema with the pruriginous dermatitides are accordingly complex, the particular cases requiring careful analysis.

Aside from these three conditions in which eczematization is extremely common, a long list of dermatoses should be mentioned which, although less frequently, may become complicated by eczema.

Mycosis Fungoides.—Mycosis fungoides includes patches of lichenoid eczema among its common initial symptoms. The exfoliating erythrodermas are often eczematized, especially in the folds. The *ichthyoses* and *hyperkeratoses* are likewise subject to eczematization. *Hyperidrosis* and *intertrigo-erythema* mustalso be mentioned among the pre-eczematous dermatoses.

ECZEMATOSIS.

Under this name I designate the chronic, constitutional or dyscratic disease, in which the eczema eruption is the principal manifestation and which is generally designated under the name of *true eczema* or *eczema disease.*

Eczematosis is met with at all ages. Certain eczemas of newborn infants belong under this heading; in the course of childhood, adolescence and youth, it seems to be an attribute of lymphatic, anemic or scrofulous individuals; at the age of maturity, it affects the overworked, inebriate and intemperate, the wealthy class being more susceptible than hospital and dispensary patients. It is especially common in arteriosclerotic and senile individuals where it becomes a very distressing and often almost incurable infirmity. I have often been put on the track of a latent visceral cancer or a partial retention of urine, pyelonephritis, etc., by incessant rebellious attacks of eczema in an aged man.

In a general way, the *etiology* of eczematosis is characterized by the fact that the local or external causes play a rather unimportant part, whereas the so-called internal causes together

with a predisposition of organic or humoral origin occupy the first rank. In the presence of eczematosis, visceral lesions and chronic intoxications are often demonstrable to such a degree that it is difficult to determine the precise cause. In other cases, the behavior suggests a sensitization of the patient through multiple or polyvalent antigens, rendering him susceptible to slight or even inappreciable causes.

Almost invariably, however, the onset of the eczematosis can be referred to a cutaneous traumatism, an accidental local irritation, repeated scratching induced by anal or genital pruritus, inflamed varicose veins, etc. Or it may take its origin in a protracted pityriasis of the scalp, or in an old patch of dry eczema. Finally, it is seen to occur, as if through the effect of a "metastasis" or substitution, at the time of spontaneous or artificial suppression of asthma, bronchial catarrh, enteritis, neuralgia or rheumatic pains.

Finally, the most trifling causes, the mildest applications, washing with soap, fatty substances and even water may provoke attacks. The skin of persons having constitutional eczema seems to have become less capable of adjusting itself to external conditions and they are attacked by paroxysms on occasions such as a change of temperature, season, climate, or humidity of the air, on exposure to wind, etc.; briefly, any condition which modifies the state of the cutaneous circulation and secretion.

The *clinical picture* of eczematosis need not detain us. It will suffice to state that the eruption may assume any form and degree, and affect any region, as discussed under eczema in general. The pruritus is variable in degree, often intolerable, paroxysmal and especially nocturnal, contributing on the one hand to the maintenance of the lesions through the scratching caused by it and on the other to the depression of the nervous system and the morale of the patient.

Notwithstanding intelligent and scrupulous *treatment,* a cure cannot always be obtained, or it is only temporary and followed by recurrences. It is very common for these recurrences or renewed attacks to start from an imperfectly extinct focus, a focus of microbic growth according to the adherents of the parasitic theory of all eczemas. The disease lasts years and sometimes till death, which may occur in consequence of the organic lesions back of the eruption, such as Bright's disease, arteriosclerosis, chronic bronchitis, cancer, diabetes, etc.

It frequently happens that the eczema disappears at the moment where the terminal complications supervene, and quite naturally so, because the exhausted organism is no longer capable of responding with a cutaneous reaction. The fact is generally interpreted

by the laity and by a number of physicians as indicating a repercussion, a "striking in" of the eruption, the effect being mistaken for the cause.

DYSIDROSIS.

Under this name, proposed by Tilbury Fox, is designated an affection (named cheiropompholyx by Hutchinson) which is generally regarded as a special disease, but which to me seems to be simply a clinical form of eczema, characterized by certain shades in the behavior of the eruption, by its seat and by its course.

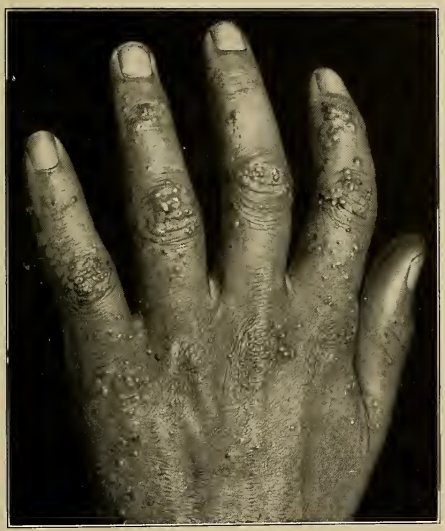


FIG. 12.—Occupational eczema of the dysidrotic types in a hair-dresser aged twenty-eight years.

Dysidrosis is seen especially in the spring, from March to June, sometimes in the fall. It recurs in a given case frequently at the same time of the year. Patients suffering from it are adults, rarely children; they present some of the disturbances referable to arthritism; frequently they are dyspeptic, nervous or overworked and perspire very readily. The attack may be caused by violent exercise or by an emotional disturbance.

The eruption begins symmetrically on the hands or feet, or all four extremities at once; it consists of small or medium-sized vesicles,

deeply set in the thick epidermis, appearing without redness but with sensations of pruritus, heat, or pain on pressure, sometimes very distressing.

It assumes its most typical aspect on the lateral surfaces of the fingers, where the skin is white or pinkish and studded with vesicles as if with grains of boiled sago. The vesicles may attain the size of a lentil and become confluent in bullæ of the dimensions of an almond or larger, especially on the palms and the soles. A clear, very stringy, neutral or alkaline fluid escapes on puncture; the fluid is sometimes turbid and on the soles is usually purulent. The vesicles of dysidrosis have not much tendency to open spontaneously and usually dry up in a few days, the epidermis becomes exfoliated and is shed, disclosing a smooth pinkish surface which does not ooze. The course of an attack lasts from five to twenty days.

The eruption may become infected, impetiginous, especially on the feet and give rise to fissures, lymphangitis, etc. It is usually symmetrical. It may invade the forearms, the neck, the face, a considerable portion of the trunk, or even become generalized when it represents what has been called a *dysidrotic eczema*.

The *pathological anatomy* of the vesicles of dysidrosis shows round or oval cavities, hollowed out at different levels of the rete. Originating in the process of spongiosis, they are filled with a moderate leukocytic fluid which causes a pressure condensation of the neighboring epithelial cells. They have no relation to the sweat channels. The special bacillus reported by Unna has not been found by others. Briefly, there are no anatomical reasons for considering dysidrosis as a special dermatosis.

Clinical arguments are of no greater value. Artificial eczema, when it affects the same localizations or any region with a thick horny layer, presents an identical aspect. Generalized dysidrotic eczema differs in no way from an ordinary eczema. Nothing definite is known as to the causes of seasonal recurrences, which are, moreover, inconstant or very irregular. Having excluded, in the case of the hands, artificial eczemas caused by turpentine, iodoform, poisonous plants or other irritants; at the feet, conditions like epidermophytosis of the toes and ordinary eczema, no cases are left entitled to the label of dysidrosis. It is therefore my belief, shared by Jadassohn, that this affection is not truly autonomous. According to Kaufmann-Wolf (1914), about one-third of the cases somewhat generally labelled as dysidrosis are of a trichophytic or epidermophytic character.

There is a desquamation of the hands and feet, a recurrent and often seasonal affection, which is referred to dysidrosis in which the epidermis dries and becomes detached at scattered points or over

polycylic surfaces. In my opinion this is connected with general nutritional disturbances, auto-intoxications, or latent infections, such as chronic appendicitis, dyspepsias, pyelonephritis, etc.

It is almost superfluous to state that dysidrosis has nothing in common with sudamina, or intracorneal vesicles observed on the trunk and limbs in cases of fever with profuse sweating; nor with hydrocystoma or intradermic cysts, due to dilatation of the sweat channels.

ACUTE DISSEMINATED ECZEMA OR MILIARY IMPETIGO.

Under this title I describe a clinical type comprising the eruptions which have been named acute disseminated morococcal eczema (Unna); miliaria rubra or alba; sudoral eruptions (prickly heat, lichen tropicus, etc.).

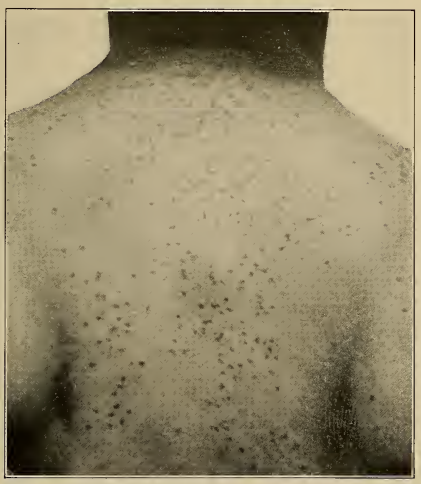


Fig. 13.—Miliary impetigo of the back, following profuse perspiration.

It is possible that this type corresponds to several distinct pathological entities; at the present time they are inseparable.

The *symptomatology* is as follows: In a young adult usually of the male sex, one notes the appearance, after profuse sweats caused by a rise of temperature, a steam-bath, hard work, or after cutaneous irritation such as a sulphur bath, of an acute

eruption of red spots, the size of a pin-head, having for their center a minute vesicle with turbid contents. There is no induration of the lesions, no papular elevation as in eczema of the papulovesicular type of Brocq; nor is the eruption follicular. It occurs especially on the trunk or the first segments of the limbs. The pruritus is variable, sometimes quitesevere.

Often, but not always, the previous existence of impetigo, impetiginized or common eczema, furuncles or a suppurating wound, can be demonstrated in these patients.

The *course* is rapid and under proper care the skin not infrequently clears up in three or four days. Successive attacks may also occur. Sometimes, especially in irritated or scratched regions one or more foci of ordinary or impetiginous eczema form.

Histology shows, at a point of congestive and edematous dermatitis, a minute blister of the impetigo type (that is, produced by cleavage), or of the eczema type (that is, through spongiosis), with serous contents more or less rich in polynuclears; sometimes there is a combination or a succession of these two processes. It is common, but *not constant*, for the little vesicle to have a sweat channel for its center, although it does not originate through dilatation of one of the canals. A follicular location is unusual.

Investigations carried out in my laboratory go to show that various staphylococci may be found in the lesions, in different cases, especially citreus and albus; these are the morococci of Unna.

In my opinion this is a microbic auto-inoculation, scattered in consequence of the maceration of the epidermis by the sweat.

It would be logical to classify this eruption with the impetigos on account of its pathogenesis and evolution, rather than with eczema, since its lesions are not agglomerated and have no tendency to oozing. I am considering it here only because objectively it resembles eczema and has no resemblance at all to ordinary impetigo.

Very simple *treatment* with non-irritating washes and applications of bland powders or watery pastes, suffices for a cure. Preëxisting suppurations should be treated if necessary, and some rest should be recommended.

CHAPTER V.

ERYTHEMATO-SQUAMOUS DERMATOSES.

There exists a group of dermatoses characterized by red and scaling spots. It might be supposed that the eruptive lesions designated by these few words are very common, but this is not the case provided the terms be used in a strict sense. The word "spots" is here employed in its widest application; by their large dimensions, the spots under consideration may be entitled to the name of patches or plaques. The *redness* must be of a congestive, erythematous character, disappearing momentarily under pressure of the finger; it is circumscribed, limited to the spots and not diffuse. *Desquamation* is present from the start and in all cases it is of the powdery furfuraceous type though sometimes micaceous or scaly and practically always connected with the modified keratinization known as *parakeratosis*.

This definition accordingly excludes: (1) Red spots which are not squamous or only become so later on; these belong to the erythemas (I); (2) Squamous spots which are not red and which belong to the *keratoses* (XI); (3) Generalized or very extensive reddening, which is known as *erythroderma* (VI).

When the spots are small and manifestly surround the hair follicles, I consider them as *folliculoses* (XIX).

Erythemato-squamous spots may be secondary to a great variety of eruptions, of erythematous, eczematous, vesicular, pustular or bullous type; representing, not a genuine eruption, but old and deformed lesions about to become *maculæ* (XVI, p. 322).

I shall here discuss only syndromes of *primary erythemato-squamous eruptions which originate and persist under this form.*

Such are: (1) The eczematides; (2) pityriasis rosea; (3) psoriasis and (4) the parapsoriases; to which will be added a few words or (5) the psoriatiform syphilides, and on (6) certain epidermomycoses.

This does not, however, exhaust the list of cutaneous affections capable of presenting the same eruptive type. As a matter of fact, it may be met with, under special features, in certain varieties of *lupus erythematodes* (XXVII, p. 568) and the *tuberculides* which approach it more or less closely; in *leprosy* (XXVII, p. 578) and in *mycosis fungoides* (XXIX, p. 657).

The reader is referred to the corresponding chapters on these diseases, which are omitted here in the interest of a more complete description. For analogous reasons, the red and squamous spots of the palmar and plantar regions, as well as those of the mucous membranes, are discussed in the chapter on the *Keratoses* (XI).

ECZEMATIDES.

I propose the name of eczematides, to designate the group of erythemato-squamous dermatoses in spots or circumscribed patches, commonly called *seborrheal eczemas*, *seborrhoids*, or *dry eczemas*.

Different authors consider them as related to pityriasis and psoriasis or as a type by themselves.

The relations of these eruptions with seborrhea are inconstant. I believe them to be closely akin to eczema, for two reasons: because of the clinical observation of imperceptible transitions and because histologically the lesions are practically identical. However, they cannot be simply incorporated with eczema, for the reason that the lesions differ clinically from the latter by the four following properties: their usual dryness; the sharpness of their rounded or polycyclic outlines; their very prolonged persistence under the same aspect; their very ready curability under the influence of local treatment.

The name I have selected seems to me convenient, indicating the affinities and suggesting the special characteristics of these eruptions.

Synonyms and Historical Data.—In view of their great frequency, the eczematides have always attracted the attention of dermatologists. They have borne very many names, which prove the difficulties encountered in their classification: *lichen circumscriptus* (Willan and Bateman); *lichen gyratus* (Cazenave and Biett); *lichen annulatus serpiginosus* (E. Wilson); *eczema acneiforme* and *pityriasis circinata* (Bazin); it is the *dry, circinate eczema figuratum* or "*flannel eczema*" of the dermatologist of the Saint Louis Hospital, the *eczema marginatum* of Pick, Köbner, Hebra and Hardy, the *seborrhea corporis* of Duhring, the *eczema seborrhœicum* of Unna. There has always been a tendency to consider these eruptions as of parasitic origin.

The name seborrheal eczema has proved decidedly the most popular. Unna, as far back as 1887, not questioning the eczematous character of the dermatosis thus named by him, pointed out that the eruption preferably attacks the seats of election of seborrhea and that, moreover, its scales or crusts have a fatty consistence. The name coined by him was based on these observations. Being struck, furthermore, by the fact that all intermediary degrees are encountered between these eczemas with fatty scales and pityriasis on one hand and certain forms of psoriasis on the other, he was led

to extend immoderately the limits of seborrheal eczema. His teachings, originally received with high favor, did not fail to arouse severe criticism. On the one hand, the eczematous nature of the process was contested, hence the name of *seborrhoids*, proposed by Brocq and Audry; on the other hand, it was demonstrated that this process is not invariably seborrheal.

The group has now been arbitrarily broken up: various constituents are classified as steatoid pityriasis, by Sabouraud, as medio-thoracic dermatosis and psoriatiform keratosis, by Brocq, etc.

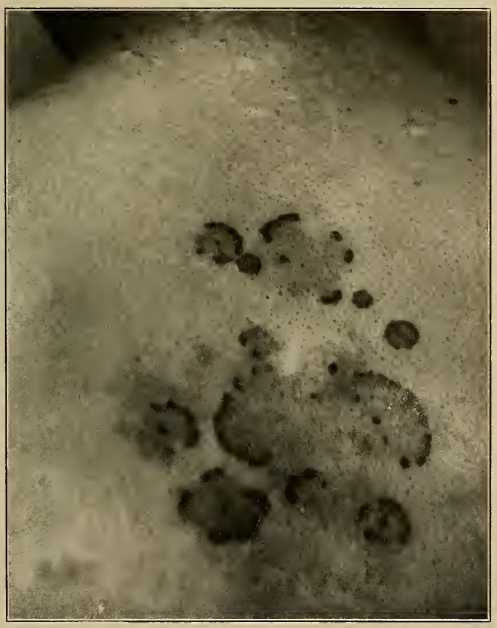


Fig. 14.—Figured eczematide of the interscapular region of the back.

In my own opinion, the prevailing confusion on this subject would practically disappear by keeping in mind: (1) that there exists a cutaneous dystrophy which I have named *kerosis* (XI), ordinarily manifesting itself through pityriasis and seborrhea; (2) that this kerotic soil is a seat of predilection for the development of various inflammatory complications which very frequently assume the

clinical form of eczematides or sometimes that of moist eczema, or still other forms, such as rosacea, acne, etc.

The eczematides are therefore interpreted by me as dry and circumscribed eczemas, which on account of these characteristics deserve to be distinguished from the ordinary eczemas, and which in the vast majority of cases, though not invariably, develop on a kerotic soil.

FIG. 15.—Figured eczematide of the forehead (corona seborrhoica, not to be confused with the "corona veneris").

Symptoms.—The eczematides are practically always preceded and accompanied in their vicinity by manifestations of kerosis. In the mildest degree, it is very difficult to discern if the complication exists or not; a judgment must be based on the presence of *circumscribed and scaly redness.*

Well-developed eczematides possess typical characteristics, differing slightly according to their seat and variety.

1. *Figured Eczematides.*—This first group corresponds to "eczéma flanellaire," the petaloid and circumscribed type of Unna, the steatoid pityriasis of Sabouraud, the medio-thoracic figured dermatosis of Brocq, the seborrhea corporis of Duhring.

The eruption at the onset almost invariably occupies the presternal and interscapular regions; from here it extends to a greater or less distance from the median line according to its severity and duration. It is seen also on the scalp, whence it extends to the forehead, temples and postauricular region.

The lesions begin as pinkish punctiform spots covered with a greasy scale. Next, they spread as nummular, petaloid or polycyclic, coalescent, more or less numerous spots, having the following characteristics: sharply marked outlines; pinkish or bright red, often slightly elevated papular borders, covered with yellowish scales and crusts which are of a fatty consistence when squeezed between two fingers; a flattened surface, on a level with the normal skin, of a yellowish pink color, often covered with scales less thick than on the margins; no apparent vesicles and no infiltration of the base. Scraping the margin with the finger-nail or with a curette causes the appearance, according to Brocq, of a minute purpura, droplets of serous fluid and, finally, small punctiform hemorrhages.

While extending, some of the spots heal in the center or in a portion of their periphery. In the zone of invasion, the follicular orifices are often attacked first, which explains the name of acneiform eczema, proposed by Bazin. Pruritus is moderate and intermittent or it may be altogether absent.

The duration of the eruption is indefinite and patients are not uncommonly met with who have suffered from it for twelve or fifteen years. Appropriate treatment causes it to disappear in ten to fifteen days.

2. *Pityriasiform Eczematides.*—The characteristic features of these cases are less definite. The lesions consist of pinkish or yellowish pink spots, with a surface covered with fine scales, dry or slightly oily in the kerotic areas and rather distinctly circumscribed. Their form is round, oval, or irregular; their dimensions and number are very variable.

The lesions may occur anywhere, but especially on the scalp and the neighboring skin, on the neck, the upper part of the trunk, the axillæ, the groins, the articular folds in general and more rarely on the extremities.

Sometimes the eruption is profuse, more or less symmetrically arranged, appearing in rather rapid attacks and composed of lenticular, nummular, or more extensive plaques, usually marginate, dotting the thorax and the abdomen. Up to a certain point, this variety can be recognized in the description of circinate and marginate pityriasis (Vidal), of *herpes tonsurans maculosus* (Hebra, Kaposi) and of psoriatiform parakeratosis in scattered patches (Brocq). It approximates the *pityriasis rosea* of Gibert. In the absence of treatment, its duration is from a few weeks to several months.

In other cases the lesions are not numerous, localized without symmetry, limited for instance to a large articular fold, developing

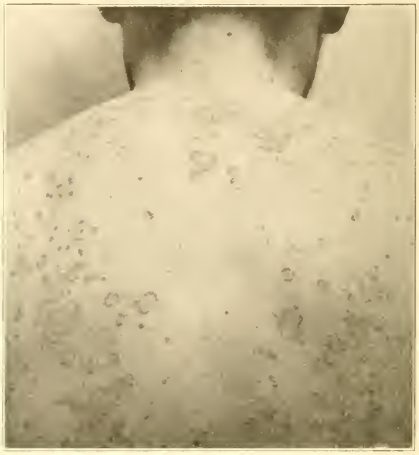


FIG. 16.—Pityriasiform eczematides.

FIG. 17.—Pityriasiform eczematide, with a lichenoid tendency, located on the posterior border of the right axilla in a young woman.

insidiously, gradually acquiring a rather considerable extent and persisting indefinitely. The spots, from being at first simply congestive and furfuraceous, may assume a lichenoid appearance, or

pass into a psoriatiform type; they are apt to present a marginate or circinate outline with a yellowish center.

In both cases, but especially in the localized variety, the pityriasiform eczematides may become temporarily vesicular or oozing (eczematized seborrhoids of some authors); then they subside again, unless they continue to develop into moist eczema.

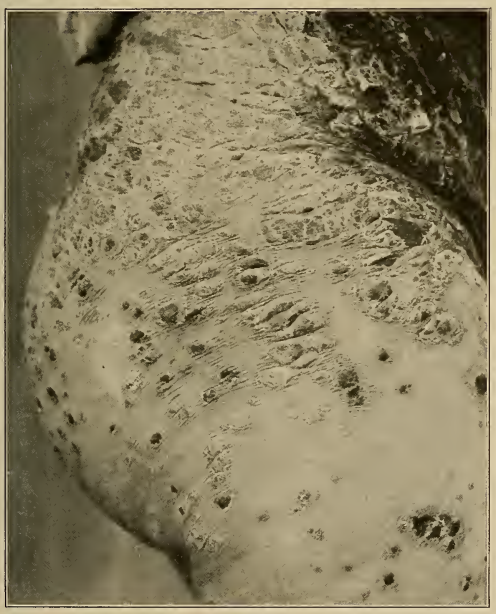


FIG. 18.—Psoriatiform eczematides on the hip of a middle-aged woman; note that on the trunk the lesions become confluent, as erythrodermia.

3. *Psoriatiform Eczematides.*—Their topography and the configuration of the lesions are entirely analogous to the preceding form. But the spots have a brighter red, or tawny hue with a slightly infiltrated base, profusely covered with more or less adherent white scales. When subjected to the systematic scraping[1] of Brocq, they yield, however, less numerous and less stratified lamellæ than the

[1] The "grattage méthodique" of Brocq consist in the careful scratching of the surface of a lesion with the aid of a dull curette; the resulting oozing, bleeding, purpura, etc., vary in a characteristic manner in different diseases.—ED.

spots of psoriasis; the red surface, on exposure, is slightly irregular and scattered over with purpuric spots, fine hemorrhages, and cup-shaped depressions from which a little serous fluid exudes, these depressions representing a pathognomonic feature. The lesions of a psoriatiform eczematide may be fairly abundant, covering a considerable portion of the integument (Fig. 18) and even becoming regionally confluent in large erythrodermic patches, covered with scaly crusts; often they are few in number, or there may be only a single large patch on any part of the body; for instance, on the neck, in a fold, or on the calf of the leg.

In this variety, moist eczematization with formation of vesicles, oozing and even impetiginization or lichenization is not uncommon.

Itching may be very severe, a symptom which may lead to possible confusion with a partial prurigo.

There exists finally, (4) a *peripilar type* and (5) an *erythrodermic type* of eczematides, which will be discussed in the chapter dealing with the folliculoses (p. 395) and the erythrodermas (p. 120).

Diagnosis.—This is by no means easy in all cases. A little attention will suffice, however, in the case of *figured eczematides*, to avoid confusion with a desiccated impetigo, with circinate syphilides, with lupus erythematodes, etc.

Pityriasiform eczematides are distinguished from pityriasis rosea of Gibert by the often larger dimensions of their lesions and by the less systematic evolution of the eruption. There are borderline cases, however, in which the question arises if a legitimate distinction can be drawn between the two types of disease. Other cases suggest a cutaneous trichophytosis; the latter is characterized by the perfect roundness of the spots, by their marginal vesiculation and by the demonstration of the mycelium on microscopical examination of the scales. Syphilitic roseola is never scaly. In psoriasis, even when mild, the scales are more profuse.

The variety with large patches is suggestive of intertrigo-erythema, of eczema marginatum of Hebra and of erythrasma; it simulates especially a circumscribed eczematized prurigo. When it presents oozing, crusts, lichenization and many erosions, the diagnosis can sometimes not be made until after treatment for several days.

Between the *psoriatiform eczematides* and genuine psoriasis, there is no definite objective difference; the distinctive features are based on the topography of the eruption, its course, its tendency to oozing and on the effect of treatment; the diagnosis in a given case must sometimes be held in abeyance.

Pathological Anatomy.—Although somewhat dissimilar at first sight, the histological lesions of the eczematides are always of the same kind, no matter what variety be studied, and all transition forms are met with between these varieties.

The essential lesions are: *Spongiosis,* in small foci; *parakeratosis,* often discontinuous; *scaly crusts; acanthosis;* in the cutis a little *edema* with perivascular *infiltration.*

Fig. 19.—Section of figured eczematide. × 27. The central portion of the nummular spot is not shown in the sketch, only the two borders of the lesion being represented. *A*, spongiosis; *B*, crust; *C*, parakeratosis; *D*, acanthosis; *E*, crust; *F*, spongiosis.

In the figured form (Fig. 19), provided the lesion is in active progression and not at a stationary stage, as frequently happens, very minute foci of spongiosis, too small to constitute vesicles visible to the unaided eye may be seen near the borders or more rarely in the center. Originating in the rete, these foci are pushed up in the course of the epidermal development as far as the horny layer, when their desiccated plasma mixed with the layers of para-

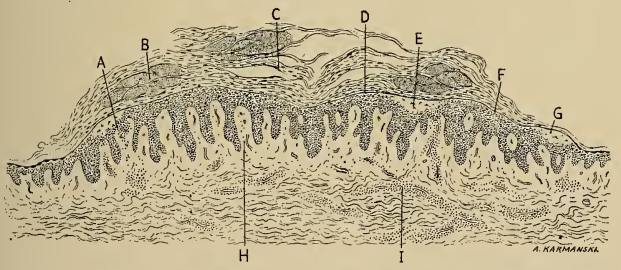


Fig. 20.—Psoriatiform eczematide. Entire section of a very small lesion. × 30. *A*, spongiosis; *B*, focus of desiccated spongiosis; *C*, scale crust; *D*, parakeratosis; *E*, spongiosis; *F*, parakeratosis; *G*, spongiosis; *H*, papillary edema and congestion; *I*, cellular infiltration.

keratotic cells gives rise to small lenticular crusts; the latter, in spite of their oily consistence, contain less fat demonstrable by osmic acid than the normal horny layer. In the center of the spots only a low degree of acanthosis is found. Edema of the papillary body and perivascular cellular infiltration are marked only near the borders, which are abrupt or gently sloping.

The psoriatiform variety simulates psoriasis, histologically (Fig.

20) as well as clinically. The differences are as follows: The superficial layer is a thick scaly crust, in which are found, between the layers of nucleated horny cells (scales), collections of desiccated serum and leukocytes (crusts). The parakeratosis may be continuous over the entire lesions, or interrupted. The acanthosis and lengthening of the papillæ are nearly as marked, although less regular than in psoriasis; the crests of the papillæ do not reach so near to the horny layer. In the papillary body very small foci of spongiosis may be discovered here and there, corresponding to a more abundant cellular infiltration in the adjacent papillæ. These are the foci of spongiosis which are destined to become crusts on reaching the horny layer. The papillary edema and the perivascular cuffs are a little more pronounced than in psoriasis (Fig. 23).

Summarizing, these lesions constitute an epidermo-dermatitis of eczema type, but more discrete, less acute and less edematous. It will be readily understood that the exaggeration of the process will result in eczema vulgare.

Etiology.—Eczematides are extremely common. They are observed at all ages, especially in the course of adolescence, youth and maturity. Many persons suffer from the affection without attaching importance to it. A considerable number of eczemas are derived from, or begin under this form.

Clinical analysis had formerly led to the conclusion that the dry figured eczemas are *parasitic*. According to Unna, seborrheal eczema is due to morococci, micrococci found in the crusts in mulberry-shaped clusters. Others believe that the seborrhea provokes eczema (Dubreuilh), or creates the necessary soil, hence the name of *seborrhoids*. Audry and Brocq, who proposed the last denomination, have demonstrated, however, that these eruptions can exist without seborrhea.

The relations between the eczematides and kerosis have been discussed above. In my opinion, kerosis creates a peculiar predisposition for the attenuated eczematization which constitutes the eczematides. In all probability, the complication is the result of a local microbic infection. The responsible species is not yet known, whether it be the staphylococcus cutis communis or the polymorphous cocens with gray cultures (Sabouraud), or other staphylococci; nor do we know if a certain degree of virulence is essential, or a certain symbiosis, for example, with the spores of Malassez (bottle-bacillus of Unna, microsporon anomæon of Vidal), or if there exists a specific microörganism which has so far escaped detection. Recent investigations of Du Bois (Geneva) on this subject indicate that the scale of the young lesions of pityriasiform eczematides—which can always be lifted as a whole with a scarifier or a fine bistoury—bears on its deep aspect collections of round

spores, of different dimensions and without mycelium; he was unable to grow cultures of this parasite or to inoculate the germ, which is probably identical with Vidal's *microsporon anomœon* or *dispar*. The same parasite is found in the scales of the pityriasis rosea of Gibert. The question therefore still remains open.

Treatment.—Whereas the treatment of eczema and psoriasis is difficult and often disappointing, the eczematides usually yield favorable results, easily obtained.

In the sluggish, especially the non-eczematized forms, treatment may consist of rather energetic local applications, grading the strength of the remedy according to the intensity of the lesions.

It is often advisable first to clean the spots, namely, to remove the scales or crusts. This is accomplished by means of soapy, alkaline, or sulphurous baths, by moist dressings, or more simply by washing twice daily with white soap, potash soap, or a sulphur or tar soap.

Directly afterward, or simultaneously, local agents are employed, salves, plasters and especially compound pastes, whose active substances are selected from the reducing agents: arsenic-zinc-sulphur paste or if this be thought too irritant, ichthyol paste, or glycerol preparations with ichthyol may be employed. More powerful reducing agents may also be used such as chrysarobin or pyrogallol in very small doses.

The psoriatiform eczematides sometimes offer considerable resistance to treatment. They are managed like psoriasis but with doses of progressively increasing strength, and cautiously for fear of exciting acute eczematization. Compounds of tar and sulphur, for example, and radiotherapy constitute very valuable adjuvants in difficult cases.

On the scalp, sulphur lotions are employed, with ointments containing the same active substance.

Internal treatment, by diet and medicinal agents, plays practically no part in the eczematides; or rather, it is identical with that of kerosis.

PITYRIASIS ROSEA OF GIBERT.

Pityriasis rosea of Gibert—*pityriasis maculata et circinata* of Duhring, *roseola squamosa* of Fournier, *herpes tonsurans maculosus* of Kaposi—is an erythemato-squamous dermatosis, characterized (1) by its lesions; (2) by its topographical distribution; and (3) especially by its course.

Symptoms.—The eruption, which itches severely, slightly or not at all, consists of two kinds of lesions. Some are pinkish, scaly, irregularly rounded lenticular or nummular *spots*, with not absolutely distinct margins; they may become confluent in plaques or in patches.

The others are more characteristic; known as "medallions," they are larger, almost invariably elliptical, pinkish and squamous on their slightly elevated borders and have a yellowish center where the epidermis is finely puckered as in atrophic striæ. These two kinds of lesions are present in variable proportions: the larger lesions, "medallions" may be rare and have to be looked for or they may be absent at a given moment.

Pityriasis rosea may occupy the entire trunk, the neck and the limbs, but it almost invariably spares the face above the chin, the hands and the feet. The immunity of the hairy scalp is worthy of special mention. The susceptible regions are not affected all at once, but symmetrically and nearly always in a certain order.

The *course* of this affection is one of its most peculiar features. The disease is almost definitely cyclic, so that pityriasis rosea has been described as a *pseudo-exanthema*.

Frequently—as was first pointed out by Brocq—the eruption is introduced by a primary patch, or initial plaque, occupying any portion of the trunk, neck, or limbs. It is erythemato-squamous, fairly well outlined, more or less pruritic, often circinate, and is usually misinterpreted as a patch of trichophytosis or an eczematide. Two or three such patches may be present. Judging from my personal experience, the initial plaque remains undetected, or is entirely absent, in about one-half of the cases.

From four to twenty days later, or sometimes from the start, the pinkish spots and "medallions" appear in profuse crops, first on the upper part of the thorax, on the neck and arms, then on the flanks, the abdomen and the thighs, finally on the forearms and sometimes on the legs.

The eruption is accordingly successive, progressive and descending. It is afebrile and unaccompanied by disturbances of the general health.

At the end of a month to six weeks, two and a half months at most, the spots which begin to fade in the regions first affected, disappear completely without leaving a trace behind.

I have observed a recurrence of pityriasis rosea at the end of four years in the same patient, but this is very exceptional.

Pathological Anatomy.—The pathological anatomy reveals more lesions than might have been anticipated. Aside from a congestion of the papillary body, with edema and rather marked perivascular cellular infiltration, Sabouraud has pointed out the regular presence in the border of the spots, of microscopical foci of spongiosis and numerous superficial histological vesicles. The latter are never clinically demonstrable and contain only mononuclear leukocytes, but apparently no microörganisms.

The scales are parakeratotic and interspersed with these desiccated vesicles. No mycelium or special parasites are demonstrable and a remarkable confusion has led Hebra and Kaposi to describe pityriasis rosea as a trichophytosis, under the name of *herpes tonsurans maculosus*.

Diagnosis.—Pityriasis rosea of Gibert differs from the eczematides, by its "medallions," by its symmetrical distribution, by its invariable absence from the scalp, and especially by its regular and cyclic development, which terminates in spontaneous cure in a definite time—though sometimes, the immediate diagnosis must be left open; from *psoriasis*, by its much less vivid redness, its fine non-stratified scales and its course; from toxic and infectious *erythemas*, by its "medallions" and its desquamation.

An unpardonable error, one that may lead to great domestic distress, is unfortunately sometimes committed, when a case of pityriasis rosea is mistaken for a *syphilitic roseola;* the latter is *never scaly*, does not form "medallions" and is associated with a hard chancre, mucous patches or at least with glandular enlargement.

Etiology and Character.—As to its nature and cause, pityriasis rosea remains a problem. It is known preferably to attack youthful individuals, especially young girls and women; to be more common in the spring and fall and in persons suffering from digestive disturbances. It shows no predilection for cases of kerosis.

Its initial plaque, its course, its spontaneous cure after a certain lapse of time and its non-recurrence, speak in favor of a systemic infection. No instances of contagion are known. Long ago, Lassar advanced the view that pityriasis rosea might result from an exogenous cutaneous infection, the unknown germ being transmitted by new or bleached underwear; Jadassohn believes this view to be justified in a number of the cases.

Treatment.—It is better to omit all treatment of an eruption which always heals spontaneously than to aggravate it by the use of soap, sulphur baths, or irritative topical applications which congest the skin, aggravate the pruritus and sometimes give rise to a real eczematization.

Dusting with a bland powder may suffice. It has seemed to me that coal-tar in small doses, particularly ichthyol, 2 per cent. in a cream or a glycerol, visibly hastened the fading of the lesions. Jadassohn recommends chrysarobin pastes of very weak concentration (1 in 3000 to 1 in 1000); others prefer sulphur pastes, or especially aqueous pastes. [Calamine lotion with the addition of 10 per cent. of precipitated sulphur, or, in very pruritic cases, of 2 per cent. of phenol is a satisfactory application.]

PSORIASIS.

Psoriasis is one of the most important dermatoses, through its frequency, the multiplicity and extent of its lesions and its rebellious character.

Symptoms.—The lesion of psoriasis is typical. In its most common form, it is a bright red, sharply circumscribed spot, covered with dry, nacreous, laminated, friable and abundant scales; the base of the spot is not infiltrated and there is no itching (Fig. 21).

Scratching these lesions causes two characteristic signs: (1) on scratching with the fingernail, the scale breaks down into a fine white micaceous dust; (2) after the scale has been removed by the fingernail a red shining surface is exposed showing fine punctiform hemorrhages.

FIG. 21.—Nummular psoriasis on the wrist, large plaque on the forearm, with psoriatic arthropathies of the hand and fingers.

The last-named sign, known as Auspitz' sign, the importance of which was shown by Hebra and Devergie, has been more fully analyzed and worked out by L. Duncan Bulkley and by the recent investigations of Brocq. It will be again referred to later.

It is frequently demonstrable, on careful inspection, that the psoriasis spots are surrounded by a pale halo, from 4 to 8 mm. wide, the skin of which is healthy, but slightly blanched.

The lesions of psoriasis are usually rounded or oval in shape. They may be of any size from a pin-point or pin-head, a drop of wax, or a coin, to immense patches, covering an entire region of the body. As a rule the lesions of the same eruption have rather uniform dimensions, hence the terms *psoriasis punctata*, [*guttata*], *nummularis, discoides*, etc., which have merely a descriptive value.

Sometimes, the lesions have the shape of psoriatic rings, from ½ to 1 cm. wide, enclosing an area of healthy skin, or fragments of

rings and arabesque designs, constituting psoriasis gyrata or figurata, formerly described under the objectionable name of lepra vulgaris; or they may be marginate plaques, the result of healing from the centre.

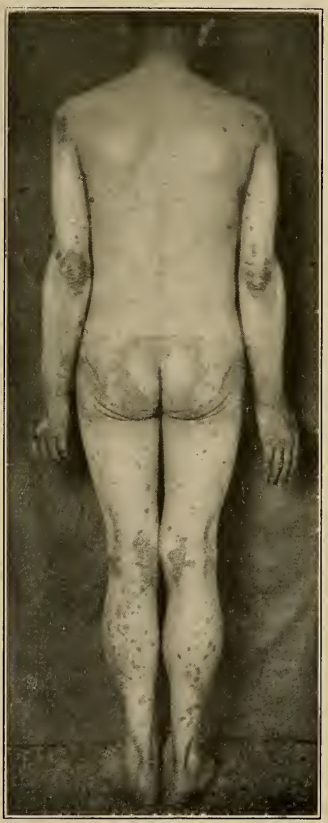


Fig. 22.—Psoriasis, general topography of the eruption.

The *eruption* is accompanied by no general disturbances; psoriasis is pruritic only in inebriates and in some nervous subjects.

The number of lesions is infinitely variable, from a few isolated spots to several hundreds. The eruption at its origin appears either

as minute red papules which fade even on scratching, or as concentrically growing guttate lesions which rather rapidly reach their permanent dimensions, or primarily as patches of nummular size.

In its *distribution*, typical psoriasis shows a marked tendency to symmetry and a predilection for bony prominences (Fig. 22); the elbows and the knees, the scalp and the sacrum (sacral plaque) are especially the seats of the largest, most typical and most obstinate patches. However, all regions of the integument including the face, the palms and the soles, the red border of the lips, and the semimucosæ of the genital organs may be affected, but the mucous membranes are always free. The "buccal psoriasis" of the older authors is simply leukoplakia (p. 219).

On the scalp, which rarely escapes and is sometimes the sole seat of the affection, the psoriasis spots or patches are characterized by their sharp limitation, the abundance of white or grayish scales, in a continuous or micaceous layer, covering a red non-oozing surface. It is noteworthy that the hairs are preserved, appear dry, pass through the scales and do not come out on traction, contrary to what happens in the eczematides of this region and in the tineas.

Varieties.—There occur anomalies of eruptive form or of distribution and regional varieties. Sometimes the spots of psoriasis are faintly outlined, superficial, light pink and covered with not very profuse slightly yellowish scales resembling pityriasis. This attenuated psoriasis, which is often seen on the face and occasionally on the genital organs, is sometimes difficult to distinguish from the eczematides.

Psoriasis spots, even when small, may exceptionally have a slightly infiltrated base, solid to the touch and almost papular; representing *infiltrated psoriasis*. This modification occurs especially at the border of the annular and carcinate lesions of figured psoriasis. Somewhat extensive spots or patches of long-standing psoriasis are always more or less thickened and sometimes even lichenoid: *psoriasis inveterata*. In certain regions, for example, on the legs, the surface exceptionally becomes roughened or *papillomatous*. Frequently, old and figured patches are covered with a nacreous, oystershell-like keratotic layer of stratified and adherent scales.

In other cases, especially in the articular folds, psoriasis may have the appearance of vivid red plaques, denuded of scales, or covered with crusts, or freely oozing like an eczema rubrum; the borders are sometimes irregular. The nature of this *oozing psoriasis*, or *eczema-psoriasis* of the old authors, is doubtful. Are these cases a special form of psoriasis, or are they an eczematized psoriasis, even though the integument of such patients is usually very rebellious to artificial eczematization; or are they a psoriatiform eczematide? It seems probable that there are cases to which each of these interpretations

may be applied. It is not uncommon to find typical spots of psoriasis on the elbows and knees of these patient.

In some cases of genuine psoriasis, the almost exclusive localization on the flexor surfaces of the articular folds, the groins, axillæ, as well as on the genitals and often on the palms and soles, constitute a type known as *psoriasis inversa*. The eruption assumes the form of large bright red patches, the skin being smooth and tense. It is found in the victims of diabetes, overexertion or intoxication. It is often very obstinate and apt to become irritated by any form of medication.

This variety more often than the typical forms, becomes associated with secondary *erythrodermas* of the benign or malignant "herpetide" type (p. 122); sometimes, the onset of this complication is attributable to badly tolerated medicinal applications.

The distribution which is indicated in the names of *palmar* and *plantar psoriasis* (p. 215); *ungual psoriasis* (p. 435) and *psoriasis universalis* (p. 120), will be described in other chapters.

A very important clinical form because undoubtedly the most *serious*, is the *arthropathic psoriasis*, studied especially by Bourdillon.

The frequency of articular manifestations in psoriasis is estimated at 5 per cent. of the cases; and in my opinion this estimate is too low. [They are by no means so frequent in America]. Often, these are merely arthralgias, myalgias and melalgias, resembling indefinite subacute rheumatism; this is *psoriasis dolorosa*. In other cases, either dating from the first eruption or after the lapse of a few years, progressive nodular, bony or fibrous arthropathies supervene, affecting the joints of several fingers (Fig. 21), one or more large joints and sometimes entire limbs and the vertebral column, leading to deformities with ankylosis and contractions which constitute most painful and deplorable infirmities.

The differential feature of these arthropathies from those of multiple arthritis deformans—with which they have many analogies—aside from their coincidence with the eruption, is their predilection for the male sex, youths and adults rather than old men, their more abrupt course, the frequent presence of hydrarthroses and the predominance of swelling among the deformities; finally, the absence of all regularity of the invasion. These features likewise characterize the tuberculous pseudo-rheumatism of Poncet.

Arthropathic psoriasis, being too common to admit of an explanation by simple coincidence, constitutes an argument for certain authors in favor of a nervous origin, for others (Audry, Petges, etc.) in favor of the tuberculous origin of psoriasis, or rather of certain forms of psoriasis.

Course.—Psoriasis runs its course in more or less abrupt attacks in different cases and is of very variable duration. The first attack

is often more acute and composed of many small lesions, but this is not an absolute rule. Subsequent attacks supervene without any regularity and often in the absence of a demonstrable determining cause.

In the interval between attacks, especially under the influence of appropriate treatment, the patient is "bleached," in the sense that the lesions fade, sometimes leaving a dark deep-pigmented macule which persists a few months before it disappears. Such patients, however, are by no means to be considered as cured.

Sometimes, two or three more obstinate spots resist all therapeutic efforts and persist indefinitely. It is moreover in the nature of the disease to recur during almost the entire lifetime of the patient. The skin rarely remains perfectly free for more than two or three years in these cases. There are innumerable cases of discrete psoriasis, without severe attacks, but incessantly persistent or recurrent.

In aged persons, the disease becomes attenuated or extinct, sometimes leaving behind it a sort of persistent pityriasic desquamation.

Diagnosis.—This is based on the characteristic features of the lesions, those of the eruption and of the disease. The properties belonging to the psoriasis spot cannot be over-emphasized. Their recapitulation here is unnecessary, but on account of their great importance, I must refer again to the data furnished by *systematic grattage* according to the method of Brocq.

In a typical lesion of psoriasis, under the successive layers of micaceous scales, a red and smooth surface is reached, from which a fine pellicle can be detached, coming off in shreds several centimeters square; underneath it fine droplets of blood exude, sometimes after a little lapse of time; but purpuric spots are rare and no drops of serous fluid are obtained, as in the eczematides.

As regards the eruption, without referring to its customary distribution, it is to be noted that in psoriasis, *all the lesions are psoriatiform*, a condition which is not found in the diseases which simulate it (psoriatiform syphilides, etc.).

It would be superfluous to dwell on the differential features distinguishing psoriasis from lichen planus, lichen corneus, premycotic patches, lupus erythematodes, pityriasis rubra pilaris and the other erythemato-squamous dermatoses treated in this chapter; these differences will be manifest from the description of these various affections.

Pathological Anatomy.—The principal lesion of psoriasis is parakeratosis, or change of keratinization characterized by the disappearance of the stratum granulosum and by the persistence of flattened nuclei in the cells of the horny layer. The horny layer is also less oily than in the normal state, its stratifications accumulate, but are easily split into lamellæ (Fig. 23).

The rete is hypertrophied and considerably thickened (acanthosis) between the papillæ which are greatly elongated and cylindrical (papillomatosis); but it is thinned above the crests of these papillæ which are separated from the parakeratotic horny layer only by a few layers of flattened cells. The papillæ and the papillary body are slightly edematous and their bloodvessels are dilated and surrounded by a small number of round cells; the lesions of the derma are insignificant.

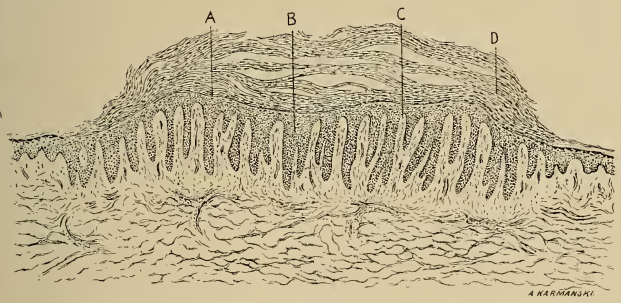


FIG. 23.—Histology of psoriasis. General section of a small but long standing lesion of the elbow. × 50. *A*, parakeratosis; *B*, acanthosis; *C*, papillomatosis; *D*, scales.

These changes explain the clinical symptoms; the redness is referable to the congestion, to the elevation of the papillary crests and to the absence of an opaque keratohyaline layer. The desquamation and the peculiar powdery scaling evident upon scratching depend upon the cleavage and the friability of the parakeratotic horny layer. The smooth sub-squamous membrane is made up of the layers of flattened Malpighian cells; it is due to the slight thickness of this layer and its softness that the adjacent vascular papillæ are easily injured with the nails, whence the punctiform hemorrhages. Acanthosis and infiltration are more marked in inveterate psoriasis with more or less lichenoid patches.

The parakeratosis, owing to which the epidermis is now composed of only two layers, like that, for instance, of orificial mucosa, is not specific for psoriasis. It is the substratum of many forms of desquamation especially of the psoriatiform type; it is observed, for example, in the dermatitides, in recent cicatrices, after the spongiosis of eczema and the eczematides, in the psoriatiform syphilides, etc.

In psoriasis itself, it is apparently only secondary. The investigations of Munro (1898) and of Sabouraud, confirmed by those of Paul Haslund (1913) have shown that aside from the lesions of the

stationary stage which I have described, small *miliary abscesses* containing leukocytes but without demonstrable microbes, may be observed in incipient or progressing psoriasis; these minute abscesses form in the subcorneal layer of the rete and are successively pushed up into the horny layer where they appear as a layer of desiccated cells. When the eruption is actively progressing, they are very numerous and succeed one another rapidly and incessantly at the same point or at neighboring points. This lesion is considered by Sabouraud as pathognomonic of psoriasis and, reasoning on these premises, he groups under the heading of psoriasis more than half of the seborrhoids or eczematides. The minute abscesses of psoriasis actually bear a close resemblance to the small foci of spongiosis which I have emphasized in the eczematides; when the two forms of microscopic lesions are confused, the histological differentiation becomes very difficult and in certain cases must rest on a single characteristic, namely the extreme paucity of microbes taking stains in sections of psoriasis, whereas there is an abundance of organisms in the eczematides.

Etiology and Character.—Psoriasis occurs in all countries and climates, somewhat more frequently in men. It may appear at any age, from two to eighty years, but is most common around the period of puberty and during adolescence. [It constitutes from 3 to 5 per cent. of all cases seen in dermatological practice.] Psoriasis is not contagious. It seems to be hereditary, or familial, in one-fifteenth to one-twentieth of the cases.

In favor of its external parasitic origin have been urged the sharp limitation and the centrifugal development of its lesions; its occurrence, in psoriatics, in localities traumatized by vaccination, tattooing, friction of suspenders or other tight articles of clothing, scratches made with a pin; a feature as a matter of fact which suggests autoinoculation. Destot (Lyons) is said to have successfully inoculated himself with psoriasis, but his experiment does not appear convincing. Arguments may also be based on the therapeutic action of parasiticidal topical applications. But the direct examination of the minute abscesses invariably shows them to be free from microbes and in the scales the common microbes are extremely rare. In the studies on the etiology of psoriasis directed by J. F. Schamberg (1913) no special microörganism in the skin or the blood of psoriatics was found. The parasitic theory, although reasonable, is by no means proved.

The relations of psoriasis with arthritism and with the dyscrasias rest on a few facts, but are neither accurate nor demonstrable. To describe a tendency to psoriasis as herpetism, is merely a play on words.

Too rich a diet, especially in albuminoid substances, seems to be

capable of favoring the first appearance, or at any rate the eruptive outbreaks, of psoriasis.

The theory of the nervous origin of psoriasis is supported by the usual symmetry of the eruption, its occasional appearance in connection with a serious accident or emotional shock, the coëxistence of neuralgias and arthropathies. It has been claimed by several observers that psoriasis manifested itself relatively often in the participants of the late war. If this be true (which is doubtful), a number of other causes besides emotion might be involved (too much meat, etc.)

Of recent years, several authors have advanced the view of a possible connection between psoriasis, or certain forms of psoriasis, and attenuated tuberculosis. Although it is certain that the arthropathies of psoriasis present marked analogies with the tuberculous rheumatism of Poncet, the great majority of psoriatics do not convey the impression of being the subjects of tuberculosis. Psoriasis and tuberculosis are so common, moreover, that there is nothing remarkable about their frequent coincidence. Brocq groups psoriasis under his "cutaneous reactions," and admits that it may develop under the influence of a number of occasional causes, or without this influence through the effect of disturbances of the general conditions not yet determined by chemical analysis. These indefinite phrases aptly express our ignorance concerning the true character of this dermatosis.

Treatment.—This must be in the first place *external* and local. It comprises two stages:

1. *Cleansing.*—It is indispensable to clean and free the patches from their scaly covering before the medicinal agents are made to act upon them. This is accomplished by more or less prolonged soapy, alkaline, or tar baths, or steam baths, or by moist or rubberized dressings. In most cases vaseline or glycerol inunctions, soft soap, lard, etc., are used. Salicylated vaseline (5 per cent.) with applications of green soap twice daily, seems to be the most rapid procedure; at the end of a few days, we may pass on to other remedies.

2. *Reducing Agents.*—All reducing agents (see Therapeutic Notes) are capable of healing the patches of psoriasis. Treatment is begun with the weakest, with the tars, especially oil of cade the virtues of which have long been tried, in the form of salves, glycerolated, or in the pure state. More or less deodorized and decolorized extracts have been prepared, known as lenicade, oxycade, etc., which are liked by patients [but are less effective].

The mercurials (calomel, turpeth mineral, etc.) or sulphur, etc., are reserved for the scalp and the face. The addition of salicylic acid (1 per cent.) increases their action. It must be kept in mind

that a mixture of sulphur and a mercurial salt would dye the hair and the same remark applies to naphthol.

Strong reducing agents, or salves containing such agents, which are efficient but irritating should not be employed except in rather scanty eruptions and when the patient can be watched.

Chrysarobin is the best; it is employed as an ointment with ichthyol, or preferably as a varnish with traumaticine. It acts only when an erythema is produced, which however, must remain moderate. Alkaline baths and washes must be avoided during its use. It stains the hair and linen a violet color and sometimes gives rise to severe conjunctivitis. But it will sometimes bleach a case of psoriasis in a fortnight.

Pyrogallic acid, in salves, varnishes, or in ethereal solutions, will destroy the linen and blacken the epidermis and the hair; it is very efficacious but difficult to handle on account of the danger of poisoning when it is employed on large surfaces.

Of the innumerable chemical products extolled abroad as antipsoriatics, none seems to me worthy of being retained.

Radiotherapy—the indications of which in psoriasis I pointed out in 1898—may rapidly obliterate recent or even inveterate patches which have first been cleansed. It is only applicable, however, in circumscribed eruptions, and does not prevent recurrences. It is employed only in exceptional cases.

3. *General Treatment.*—This is much less important. Arsenic has been regarded as a specific and administered in all its forms; moderate doses are often beneficial; energetic arsenical medication subjects the patient to the risk of intense pigmentation and may produce a genuine leuko-melanodermia. The arsenobenzols are free from this drawback and are sometimes very successful but must be handled very cautiously.

Potassium iodide, in enormous doses from 15 to 25 grams daily has been advocated; I have obtained no results from it. Injections of calomel, or of yellow oxide, by themselves alone have sufficed to cure some cases of typical psoriasis and to improve markedly arthropathic psoriasis; unfortunately they are not reliable. Experiments are now under way with injections of sulphur in solution.

It goes without saying that proper hygienic measures should be recommended with abstinence from alcohol and stimulants. Schamberg and his associates (Philadelphia Research Laboratories) have emphasized a habitual nitrogen retention in these cases and the favorable influence of a vegetable diet poor in nitrogen, as previously recommended by Bulkley, Besnier and others.

Warm mineral springs and sulphur waters are useful in some cases.

PARAPSORIASIS.

Under this provisional name, Brocq in 1902 grouped several rare and unclassifiable dermatological types, characterized by non-pruriginous erythemato-squamous spots, extremely persistent and rebellious to all treatment.

It is exclusively the objective appearance of these dermatoses which led to their mutual approximation to psoriasis. Their origin and character are unknown, hardly even surmised, and probably variable.

Three types are distinguished:

Parapsoriasis Guttata.—This eruption is disseminated over the trunk and limbs in faintly pink or brownish-red lenticular spots, very slightly infiltrated, covered with dry adherent scales, comparable to a sealing wafer. Scratching of these spots gives rise to fine purpuric puncta. The eruption resembles an abortive guttate psoriasis, or a resolving papulo-squamous syphilide. It is maintained by the appearance of new lesions, originating separately or in crops.

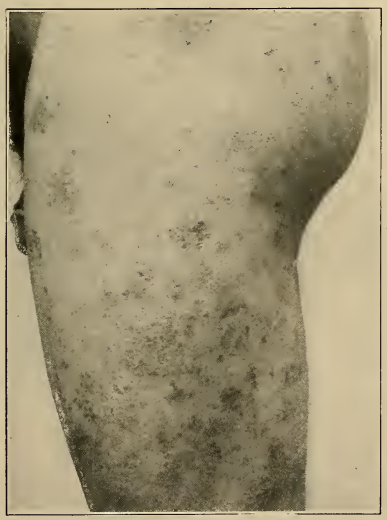


FIG. 24.—Lichenoid parapsoriasis dating back four years, in a man aged thirty-two years.

Parapsoriasis Lichenoides.—This type differs from the preceding by the more papular, more infiltrated and less psoriatiform

character of the lesions. These appear in the form of bright pink, hemispherical or flattened, glistening and non-scaly papules; later on, the lesion becomes purplish and bears a scale which seems to be imbedded in a depression of the papule; finally there remains only a yellowish macule with slight atrophy of the epidermis. The eruption is scattered over the trunk and the limbs or is arranged in clusters and network; it is not at all pruritic. The reproduction of the lesions during years in the same regions gives the skin a peculiarly mottled appearance (Fig. 24). The differential diagnosis must be made from lichen scrofulosum and other tuberculides, lichen planus, syphilides and psoriasis. The cases published by Unna [and Pollitzer], Jadassohn, Neisser, Pinkus, Crocker, etc., under the names of *parakeratosis variegata, lichen psoriasis, pityriasis lichenoides chronica*, etc., belong to the first two forms.

FIG. 25.—Parapsoriasis in patches of thirty years' standing on the flank of a man, aged forty-seven years.

Parapsoriasis in Plaques.—It consists of circumscribed spots or patches of a yellowish pink or wine color, with few or no scales, non-infiltrated and non-pruritic; their configuration is round, oval, zoniform, annular or reticulate (Fig. 25); their arrangement varies slightly from year to year; on their surface the texture of the skin is modified and the normal mosaic more marked. The eruption occupies the trunk and the limbs. It resembles the eczematides,

premycotic plaques, or tertiary syphilitic erythemas. This type was described in 1897 by Brocq, and later by J. C. White, under the name of *pityriasic erythroderma in disseminated patches.* Crocker gave this form the name of *xantho-erythroderma perstans.*

The *histology* of parapsoriasis, very carefully studied by Civatte, presents briefly the following lesions: Edema and congestion of the papillary body, with perivascular cuffs consisting especially of lymphocytes, sometimes collections suggestive of lichen scrofulosum; a rather atrophic Malpighian layer with small areas of parakeratosis.

Parapsoriasis appears at all ages, especially during youth and maturity, its duration is indefinite; the lesions disappear, while others insidiously supervene. Its nature is unknown; in some of the cases the patients were old syphilitics; on the other hand, the theory maintained by Civatte, according to which the disease is a tuberculide, is based on strong clinical and pathologico-anatomical arguments.

All local medication is ineffectual; injections of arsenical preparations and of mercury have seemed to me useful in some cases.

PSORIATIFORM SYPHILIDES.

Strictly speaking erythemato-squamous syphilides do not exist. However, as it is always in connection with syphilis that the question of the diagnosis of pityriasis rosea, psoriasis, etc., arises, and as errors are frequent, I believe the insertion of the following paragraph will be useful.

In the first place, let me point out that syphilitic roseola is never squamous or scaly (A. Fournier), whereas, pityriasis rosea and psoriasis are *always* scaly.

As to the papular syphilides of secondary lues, these are habitually slightly scaly, but may become so to a degree which entitles them to the epithet of *psoriatiform.*

Their dimensions are miliary, lenticular, or nummular; their form is round, orbicular, sometimes annular in certain regions; their color is pink or red, but often dull, purplish, not so vivid as the lesions of psoriasis. Although desquamation may be profuse, it is nevertheless not so abundant as in psoriasis; scratching reveals no subcorneal pellicle and very readily produces traumatic purpura.

But the sign par excellence of the syphilides, on which the diagnosis must always rest, is their firm resistant *infiltration,* their hardened, neoplastic consistence; they have substance, according to an expression used by A. Fournier. The lesions of recent psoriasis, on the contrary, are pliable and not indurated. All this signifies that in spite of their apparent anology, syphilis really produces squamous papules, whereas psoriasis gives rise to squamous macules.

The eruption of psoriatiform syphilides is irregularly scattered

everywhere, often confluent on the face, the back, the nape of the neck; no patches are found on the elbows, knees and scalp. The eruption is more or less polymorphous, not all the lesions are psoriatiform; finally, there are coincident glandular swellings or adenopathies, mucous patches, etc.; briefly, signs of syphilis, and the Wassermann reaction is positive.

The tubercular syphilides of tertiary lues may be abundantly squamous and psoriatiform. However, they are firm to the touch, usually circinate, always segregated, regional, not very numerous. Moreover, healing is followed by cicatrices.

ERYTHEMATO-SQUAMOUS EPIDERMO-MYCOSES.

Several parasitic cutaneous diseases, due to vegetable parasites, may assume the form of red and squamous spots.

This is rarely the case in pityriasis versicolor; its spots are yellowish or brownish; exceptionally they may be of a pink color. It is noteworthy that one of the peculiarities of microsporon furfur is that it produces practically no congestive or inflammatory reaction.

Erythrasma.—This affection on the contrary often leads to confusion, especially with the pityriasiform eczematides in large patches. It is distinguished by the dryness of its scales, by its localization, by its persistence, by the almost complete absence of pruritus, and especially by the presence of the microsporon minutissimum in large numbers in the epidermis (p. 532).

Trichophytosis of the Smooth Skin.—This is characterized by the generally perfect orbicularity of the red and squamous spots, by their relatively rapid and regular peripheral development, by the rather frequent presence, especially on their borders, of vesicles which have earned for this affection the name of *herpes circinatus* (p. 522).

Epidermophytosis.—The rapidly extensive, red and squamous spots, with polycyclic contours, bordered by small vesicles or a scaly margin, which develop in the groin and in the large cutaneous folds, constituting the *eczema marginatum* of Hebra, are due to the epidermophyton inguinale; the microscope readily reveals the mycelium of the parasite in the scales (p. 524).

Microsporosis.—Various microsporons may vegetate on the hairless skin; the spots caused by them are of irregular form, indistinctly outlined, more pityriasic than erythematous and very readily curable (p. 527).

Favus Cutaneus.—This may appear without "cups" in the form of red and squamous, rarely vesicular, distinctly outlined and fairly regular rounded spots (*favus herpeticus*); the achorion is abundantly represented in the scales.

Tropical Epidermomycoses.—These are discussed elsewhere in this book (p. 528).

CHAPTER VI.

ERYTHRODERMAS.

The name *erythroderma* is applied to a very extensive or generalized, persistent and squamous inflammatory reddening of the skin.

The symptom of extensive and persistent redness is very easily demonstrable; but as it occurs under very different pathological conditions, its significance and value vary to a high degree.

The redness of erythroderma is *inflammatory*, so that very extensive vascular nevi, for example, do not belong under this heading. The vascular congestion is sometimes accompanied by a certain degree of swelling or retraction of the tissues; the skin feels warm to the touch, but the patients frequently complain of a constant cold sensation.

The redness is *very extensive;* however, the erythrodermas are practically limited in degree to the chronic erythemas and the erythemato-squamous dermatoses.

It is *persistent.* The meaning of this word is somewhat elastic; usually, to be declared as erythrodermic, the duration of a red eruption must exceed seven days.

Finally, and especially, the erythrodermas are *squamous* from the start or after a few days, very abundantly or scantily, under variable forms, powdery, furfuraceous, lamellar or exfoliating, etc. The epithet *exfoliating* or *exfoliative* applies to a considerable number of the most characteristic types; other cases have been designated under the name of *pityriasis rubra.*

The term "pityriasis" according to Besnier, is a simple dermographic expression specifying a particular form of epidermic desquamation in lamellæ as fine as bran, or furfuraceous. It is a traditional designation, not indicative of any relationship between them, for cutaneous affections differing as widely as pityriasis simplex, pityriasis versicolor, pityriasis rosea of Gibert, pityriasis rubra pilaris. The common denomination "pityriasis rubra" therefore does not apply to all erythrodermas and is reserved in this book for the variety described by Hebra.

The symptom of erythroderma is met with under four different conditions: (1) As a constituent of peculiar eruptions developing on the healthy skin; these are the *Primary Erythrodermas.* (2) Through the effect of generalization of one of the dermatoses with

red and squamous spots, described in Chapter V; for this group I reserve the name of *Erythrodermic Dermatoses*. (3) As a complication of a preëxisting eruption; these are the *Secondary Erythrodermas*. (4) Finally, there occur *Congenital Erythrodermas* and cases developing in *newborn* infants.

I. PRIMARY ERYTHRODERMAS.

Acute forms have been described, lasting a few weeks; subacute forms, lasting several months; and chronic forms, lasting a year or longer.

This subdivision, although convenient for the grouping of observations, implies neither difference nor identity of character among these clinical forms.

A. **Primary Acute Erythroderma.**—This is a generalized erythema with a foliaceous desquamation, of toxic or infectious origin, also known under the name of *Recurrent desquamative scarlatiniform erythema* (Feréol and E. Besnier) and *Acute benign exfoliating dermatitis* (Brocq).

After two or three days of prodromata, in the form of prostration, headache, chills, and fever of 38° or 39° [100° to 102°] the eruption makes its appearance as a red pruritic surface in the large folds of the trunk and the limbs; it becomes generalized in a few days, the head sometimes escaping.

Before the redness disappears, desquamation begins and gradually extends; it is locally furfuraceous but more apt to occur in large collodium-like shreds or on the hands and feet, in the form of an incomplete glove or sandal (Fig. 26). The skin underneath appears smooth, sometimes still scaly, or oozing in the folds. The mucous membranes may be affected; redness of the conjunctivæ, an erythematous angina and desquamation of the tongue are noted. The general condition becomes practically normal long before the end of the cutaneous disease, which lasts about three weeks. The nails remain marked by a transverse furrow; loss of hair is inconsiderable. Relapses are frequent, at intervals of months or years, but usually diminish in severity.

The causes of this eruption are imperfectly understood. A necessary predisposition and various causative factors are admitted. First in order come the intoxications, notably mercury, the influence of which should be looked for in all its forms: internal, external, medicinal and accidental; picric acid, quinine, chloral, belladonna, opium, may also be responsible. When toxiderma can be excluded, infections are to be thought of, such as gonorrhea, malaria, streptococcus infection, etc.

The differential diagnosis must be made from the scarlatinoid

erythemas, which are less extensive, more transitory and less desquamative; and especially from scarlet fever which is not recurrent, is accompanied by angina and more pronounced general phenomena and has a more belated desquamation. [The most important differential feature is found in the circumstance that the exfoliation usually begins while the erythema is still pronounced; but a diagnosis is often impossible and] in doubtful cases the same prophylactic measures are called for as in the presence of scarlatina.

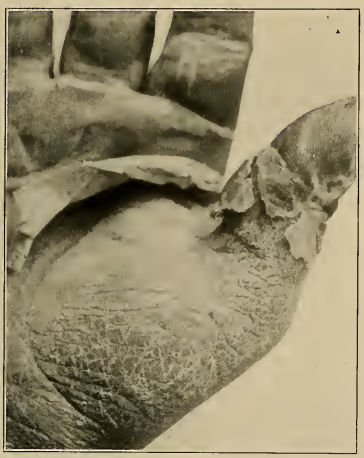


FIG. 26.—Acute primary erythroderma in the stage of desquamation following ingestion of a mixture containing opium (Bergé's case, Soc. Méd. des Hôp., February 22, 1907).

B. **Primary Subacute Erythroderma.**—This probably represents merely a more prolonged and more serious form of the preceding type; it is also known as *generalized exfoliative* or *exfoliating dermatitis* of *Wilson-Brocq*.

The onset is the same, with or without prodromata; generalization is complete, somewhat more gradually established; the foliaceous desquamation is so active that handfuls of epidermal shreds may be gathered up in the morning from the patient's bed. The mucous membranes and the appendages are always involved; the nails and nearly all hairs of the body may fall out toward the end of the third to fourth week.

The tension of the skin, the constant sensation of cold, the loss of weight notwithstanding the good appetite, the diarrhea, the great

diminution in nitrogen excretion and the hectic fever indicate the gravity of the disease. Death occurs from cachexia or as the result of complications, in one-sixth of the cases. The duration is from three months to a year.

This rather uncommon subacute form is observed in adults, especially in inebriates and in connection with some form of intoxication or auto-intoxication. Relapses hardly ever occur.

C. **Primary Chronic Erythrodermias.**—At the present writing, three types can be differentiated.

FIG. 27.—Chronic exfoliative dermatitis of three years' standing, in a man aged sixty-six years.

1. **The Chronic Form of the Exfoliative Dermatitis of Wilson-Brocq.**—Under this heading are grouped the cases analogous to primary subacute erythroderma but lasting several years (Fig. 27).

2. **Pityriasis Rubra of Hebra-Jadassohn.**—The erythema begins in various regions, especially in the large folds, as red surface lesions with furfuraceous scales, without infiltration or oozing. The complete generalization takes place in a few months, at most two years; chills are common and itching is variable.

The skin gradually becomes thickened, then atrophied and retracted to the point of interfering with movements. The appendages fall out. Death supervenes at the end of a few years, in a state of general marasmus and according to Jadassohn almost invariably through tuberculosis. Since his investigations (1892) there is a tendency to group pityriasis rubra under the heading of the *Tuberculides*.

Cases of partial eruption have been reported, as well as subacute benign cases, the interpretation of which is doubtful.

3. **Premycotic and Leukemic Erythrodermas.**—In male adults, the onset of an erythroderma has been observed beginning with highly pruritic red spots, becoming universal, scarlatinoid, more purplish in the folds and dependent portions, sometimes with depressed white spots scattered here and there. Desquamation is usually very slight or it may be dry and lamellar. Febrile attacks may occur. The nails usually remain intact, but the hairs fall out to a great extent.

Three constant features attract attention: a frenzied irrepressible pruritus, causing the nails to become worn off, edematous thickening of the skin, which forms ridges toward the great folds and generalized glandular swelling.

After a very long time, up to ten years or more, small cutaneous nodosities may make their appearance, having the structure of mycosis fungoides (p. 659).

This premycotic erythroderma, pointed out by Besnier and Hallopeau, may present prolonged remissions; it may also lead to death from cachexia before the apperance of tumors.

The *lymphoderma perniciosa* of Kaposi was probably an erythroderma of this kind, which became complicated by leukemia (p. 655).

The **leukemic erythroderma** studied by Audry, Nicolau, and others, is a pathological type closely related to the preceding. Desquamation is perhaps more profuse and pruritus less severe; but especially, in addition to the adenopathy, there is hypertrophy of the spleen and a relative or absolute leukemia. Sometimes, the appearance of small nodules in the skin, or papillomatous patches, has been noted. Death is inevitable and may supervene in less than two years.

All cases of primary chronic erythroderma necessitate, from the diagnostic and prognostic point of view, thorough clinical study with careful examination of the viscera and glands, complete and repeated examinations of the blood and sections of the skin and the glands, for histological and bacteriological examination as well as animal inoculation. Only in this way can we hope to determine the relations of pityriasis rubra and mycosis fungoides with the leukemias, pseudoleukemias, tuberculosis, etc.

II. ERYTHRODERMIC DERMATOSES.

Some of the great dermatoses may become erythrodermic through generalization in the course of their evolution. In these cases, the characteristics of the eruption persist, only modified by the extension it has undergone.

Generalized Eczema.—Generalized eczema proceeds in successive relays, invading new territories and becomes permanently established if the conditions of eczematosis exist in the patient; but it rarely becomes universal. Even in these cases it retains its tendency to recrudescences, paroxysms, oozing, to a reddened appearance and severe itching. The mucous membranes remain intact and the appendages are only gradually affected. The general condition is far from being as seriously affected as in the cases of true erythroderma, whether primary or secondary.

The principal difficulty is the recognition of the nature of this eczema, whether it is primary, or secondary to a prurigo, for instance, and on the other hand to discover the nutritional disturbances, internal suppuration or visceral lesion of which it is often a manifestation, such as nephritis, cancer, etc. The treatment is not very effective and must be very cautiously handled.

In generalized eczematide—*pityriasis rubra seborrhœica* of Unna, or malignant exfoliating form of seborrheal eczema—the eruption extends little by little but is rarely universal. The eruptive areas or widely invaded regions are sometimes red and dry, covered with pityriasic fatty scales, sometimes oozing and covered with yellowish scaly, not very adherent crusts, the contours are rounded or polycyclic (see Fig. 18, abdominal region); in the folds and under the thick crusts, serous or purulent oozing is present in all cases.

It is often difficult, until a few days or weeks of treatment have elapsed, to distinguish between a generalized eczematide, a primary or secondary eczema and an eczematized psoriasis. The rounded crusts may even suggest pemphigus. The general condition usually remains favorable. The prognosis is less gloomy than that of the other generalized dermatoses and especially that of exfoliating herpetide (p. 122). It depends upon the degree of toleration of the skin for the topical reducing agents which must here be employed with moderation.

Psoriasis universalis may finally become absolute and total; the redness is uniform from head to foot; the desquamation loses its stratified and micaceous character, except at the seats of predilection; neither oozing, nor crusts, nor pruritus is present. The hairs of the scalp and body become scanty; the nails are striated and ridged. Arthropathies may develop.

The course, without paroxysms, is of an absolutely chronic

character. Treatment often remains completely ineffective; although I have obtained nearly complete but always temporary cures by means of mercurial injections, using calomel or the yellow oxide. The arsenobenzols might be given a trial.

Pityriasis rubra pilaris may exceptionally, at the onset or in consequence of its progressive extension, present an erythrodermic appearance. But healthy skin areas can always be demonstrated, in distinctly outlined, sometimes angular islands as well as peripilar cones on the borders of the surface lesions or at the points of election. The desquamation is plaster-like and adherent.

Acute lichen planus, sometimes spreads over large surfaces and may assume an erythrodermic appearance; this abnormal extension is usually initial and rapidly retrogressive. It soon becomes possible to discover the characteristic papules, if necessary with the assistance of a lens on the red surfaces themselves or at their borders.

Pemphigus foliaceus occasionally presents to a high degree the clinical appearance of an abundantly exfoliating erythroderma. The disease has generally passed at the onset through a stage of bullous eruption and sometimes bullous lesions can be found around the erythrodermic areas. The exfoliation of pemphigus foliaceus is noteworthy for the moist or even oozing condition of the tissues underneath the scales.

Equine scabies is a very rare disease which may appear under the guise of erythroderma, as illustrated by the case of Besnier's and a personal observation of my own. The redness is universal, involving even the face and the scalp; the crusted or powdery scales predominate on the hands and feet; itching is not excessive. No burrows can be detected; but even the smallest scale is seen under the microscope to teem with the parasite (sarcoptes) in all its stages.

III. SECONDARY ERYTHRODERMAS.

Generalization, as has been seen, leads to the so-called "erythrodermic" appearance in the great dermatoses which have just been briefly reviewed. On the other hand, their course may be temporarily arrested by, or it may terminate in a very extensive or even universal exfoliating dermatitis; this marks and transforms the characteristics of the first eruption and seems to take its place, reproducing the aspect and behavior of a primary erythroderma.

In the first case, there has been an extension with more or less complete preservation of the characteristics belonging to the first eruption; the second case, where there is unification of the pathological picture, was interpreted by the older authors as a transformation, whereas modern writers are inclined to see in it the effect of a complication.

It is readily understood that an investigation of the patient's antecedents and preliminary condition is usually required in order to establish the fact that the erythroderma is really secondary and the nature of the dermatosis on the soil of which it has developed.

The secondary erythrodermas manifest themselves under two forms, ordinarily designated by the traditional names of *benign herpetide* and *malignant herpetide*.

Benign Form (episodic erythrodermas of Besnier). — The erythrodermic complication may be partial, regional or very extensive; rarely is it total; it is always transitory, lasting a few days to a few weeks. There are valid reasons for ascribing it to the inopportune or badly tolerated intervention of an external medicinal agent, such as a mercurial preparation, chrysarobin, picric acid, etc., or of some internal medication. Relapses are extremely liable to occur.

Malignant Form (exfoliative herpetide of Bazin, chronic malignant exfoliative dermatitis of Vidal and Leloir). — By Bazin, who described it, exfoliative herpetide was interpreted as the common outcome of certain eczemas, psoriasis, pityriasis; supervening by imperceptible transition and representing a sort of cachexia of the skin, comparable to asystoly in heart disease.

The symptoms are those of the subacute form of primary erythroderma, but with less fever; there is, however, well-marked general exhaustion and marasmus. The elimination of urea in the urine, always greatly diminished, may be reduced to 10 or even 4 grams per day; on the other hand, up to 10 grams of urea have been found in the daily scales.

The prognosis is fatal although more or less prolonged remissions have been observed.

There is a tendency to attribute this "malignant herpetide" to a toxic, infectious or autotoxic complication of the same character as that which gives rise to the primary erythrodermas.

IV. CONGENITAL ERYTHRODERMAS AND ERYTHRODERMAS OF THE NEWBORN.

In little children, various types of congenital or acquired, temporary or persistent exfoliative erythrodermas have been observed. The following cases must be distinguished:

1. **Lamellar Desquamation in the Newborn.**—This represents an exaggeration of the phenomenon of physiological desquamation seen in many newborn infants and consists of desiccation and splitting of the epidermis in the first days of life, followed by the shedding of this epidermis, furfuraceous or in shreds, from the third to fifth day up to the thirtieth or sixtieth, according to Parrot.

In rare cases, such as that observed by Grass and Török (1895),

the child is born with a sort of supra-epithelial collodium-like layer (*epitrichium*); this splits after the first hour and in a few days becomes detached in the form of large shreds; the skin then becomes normal again.

2. **Exfoliative Dermatitis of Nurslings.**—Described by Ritter von Rittersheim (1878) begins during the first to fifth week of life, around the mouth or, more rarely, in other parts of the body; it becomes rapidly generalized over the entire integument and finally extends to the extremities. The skin is of a purplish-red color and is desquamated in large dry shreds. Bullæ have sometimes been observed. Certain authors believe that this affection is related to epidemic pemphigus. As a rule, the disease is febrile and in one-half of the cases it leads to death, often within a week.

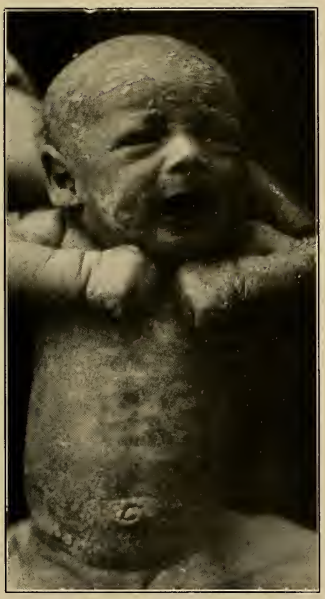


FIG. 28.—Erythroderma in a newborn infant (generalized eczematide.)

3. **Generalized Dermatoses.**—Various generalized dermatoses, including medicinal, mercurial and other eruptions, are encountered in children.

I have published a case of seborrheal eczema or eczematide in an

otherwise healthy child of five weeks (Fig. 28); onset at the ear, almost total generalization in nine days; scarlatinoid redness with dry scales or greasy crusts, in different localities; recovery in three weeks.

Desquamative Erythroderma of Nurslings, described by C. Leiner in 1907, is a morphologically analogous morbid type. He was enabled to observe 43 cases, usually beginning on the scalp, with 15 deaths. Beck's record of 16 personal cases, in which the disease usually began on the buttocks, confirms the observation that this grave eruption, which seems to be of toxic character, is peculiar to breast-fed children suffering from enteritis. A change of alimentation is urgently required.

4. **Diffuse Congenital Hyperkeratosis.**—Also known as *congenital* or *fetal ichthyosis*, or *ichthyosis sebacea* (Kaposi), is a cutaneous malformation of which several degrees are known:

The *severe type* (*diffuse congenital malignant keratoma*) [*harlequin fetus*] is incompatible with life. The infant, born at term or prematurely, presents a dreadful appearance; its entire skin is red and tense, as if too tight, fissured, covered with large yellowish layers or crusts several millimeters thick, apparently resulting from the desiccation of a sebaceous coating. The face is formless; movements of the limbs are almost impossible; the child is unable to suckle and promptly succumbs to cold.

The *benign type* (*generalized ichthyosiform hyperkeratosis*) is not fatal; it is usually confused with ichthyosis. It will be discovered in the chapter on the Keratoses (p. 203).

Pathological Anatomy of the Erythrodermas.—The lesions of all erythrodermas are associated with vascular congestion, a variable cellular infiltration in the papillary body, with more or less edema and pigmentation and corneal exfoliation, the mechanism of which usually remains obscure.

Reliable histological investigations are not numerous and have yielded divergent results in forms bearing the same label. At the present writing a differential histological diagnosis between the various types is out of the question. The following are the few available data on this subject:

In the primary subacute erythrodermas there is claimed to be parakeratosis, intrapapillary infiltration with enlargement of the papillæ. Central, ganglionic and peripheral nervous lesions have been described by Mario Oro.

In the pityriasis rubra of Hebra the papillary body, at first infiltrated, later on undergoes atrophic changes; the glands and follicles disappear; the stratum granulosum is diminished or absent. In a very remarkable case Brunsgaard found in sections of the skin typical tubercles with giant cells and Koch's bacilli. It had already

been shown by Jadassohn that in autopsies on cases of pityriasis rubra, tuberculosis was found in various organs in seven out of eight cases.

Premycotic erythroderma represents the most distinctive anatomical type. The illustration gives a fairly accurate idea of the condition (Fig. 29).

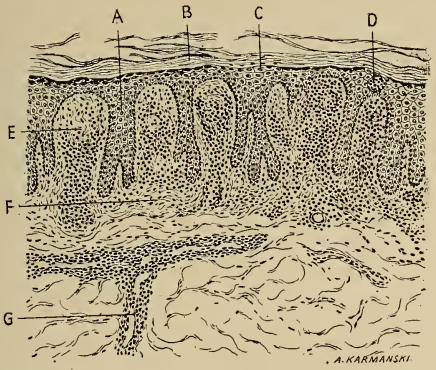


FIG. 29.—Histology of premycotic erythroderma. The dominant lesion consists of a very dense cellular infiltration (*F*), occupying the papillary body and having a sharp lower boundary; it is composed of lymphoid cells arranged in an adenoid network. The afferent vessels of the chorion (*G*) are surrounded by cellular cuffs. The papillæ (*E*) are enlarged and elongated. The interpapillary buds (*A*) are drawn out and often bifid. The horny layer (*B*) is thick and desquamating. Parakeratosis is noted in places (*C*). In the rete may be seen minute cellular nests (*D*) filled with lymphocytes; those are inconstant, but possess great diagnostic value, being specific of mycosis fungoides. × 65.

In the malignant exfoliative herpetides there exist parakeratosis, according to Leloir, and a destructive change of the connective-tissue fibers, especially around the vessels, with preservation of the elastic fibers. These lesions, which were reported as characteristic, could not be confirmed by Mario Oro.

The generalized dermatoses preserve the histological lesions peculiar to them, although with some modifications.

Treatment of the Erythrodermas.—The most essential requirement is to ascertain with great care the possible *cause* of the erythroderma. When there is reason to suspect an intoxication, or some external agent, this injurious factor must, of course, be removed. An existing auto-intoxication should be controlled by dietetic measures, free ingestion of water, enteroclysis, injections of glucose or other sera, together with general hygiene. With special reference to

mycosis fungoides and the leukemias and pseudoleukemias, radiotherapy is known to furnish a valuable method, if not for a cure at least for improvement and retardation of the course of the disease.

Local Treatment must aim especially at *non nocere*. It comprises, depending on the case, prolonged or even permanent emollient baths, such as were formerly given abroad; moist aseptic dressings, or applications of oil and lime-water liniment, which are very troublesome to handle but afford a marked relief for the pruritus; finally, wrapping in cotton. It is often useful to cover the patient [thickly and continuously] with a bland powder between two sheets and to apply lotions only here and there or partial inunctions with a paste or a cream. Caoutchouc must be very cautiously employed; it will blanch certain erythrodermas, it is true, but seems to be capable of inducing very dangerous repercussions.

All these topical agents are merely palliatives, aiming at the relief of the patient and the prevention of complications. They represent simply a marked form of expectant treatment.

CHAPTER VII.

PAPULES AND PAPULAR DERMATOSES.

The eruptive lesions described as *papules* are small solid elevations which subside spontaneously. The terms of this definition may be stated with greater precision, as follows:

The papules are circumscribed elevations of small dimensions; they have the size of a pin-head, a lentil or at most a large pea; they are always protuberant, but to a very variable degree.

The papules are solid, which means that they do not contain an effused fluid; it is sometimes necessary to prick them with a needle in order to ascertain this fact. Finally, papules do not persist indefinitely, a feature which distinguishes them from small tumors of the same appearance; they disappear of their own accord, without leaving a scar, which differentiates them from tubercles; this is expressed in the statement that they undergo spontaneous resolution.

Many papules develop exclusively at the site of the hair-follicles; these *follicular papules* will be discussed with the folliculoses (XIX).

The papular prominence may become associated with various processes, for instance hemorrhage (*papular purpura*); vesicle formation in certain eczemas (*papulo-vesicular eczema*), and formation of pustules (*papulo-necrotic tuberculides*), etc.

Very large papules are sometimes designated as *papulo-tubercles*.

It sometimes happens that an infiltration similar to that which constitutes the papules, instead of being closely restricted to a very small surface, is spread out on the contrary in coin-shaped (nummular) discs or in patches and becomes superimposed on an erythematous, erythemato-squamous, etc., process. In such cases, although these expressions are not, strictly speaking, correct, it is customary to employ the terms of *papular plaques* or *patches*.

Anatomical Characteristics of the Papules.—The eruptive lesion known as a papule may be produced through different pathological processes. According as they affect chiefly the epidermis or the derma or the two tissues alike, a distinction is made between epidermic papules, dermic papules and mixed papules.

Epidermic Papules.—Epidermic papules are most typically represented by the *flat warts* (Fig. 30). In these, all the layers of the epidermis are hypertrophied, the mucous body (acanthosis) as well

as the granular layer (granulosis) and the horny layer (keratosis). The papillæ are elongated (papillomatosis) up to ten times their normal height. There is very little edematous or cellular infiltration in the derma.

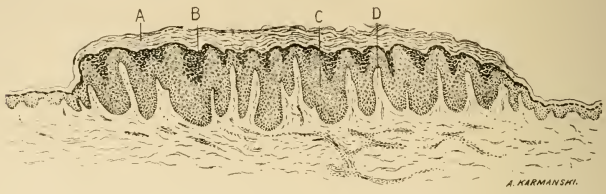


Fig. 30.—Section of an epidermic papule. Flat wart of the face. *A*, keratosis; *B*, granulosis; *C*, acanthosis; *D*, papillomatosis. × 33.

In the papule of prurigo, the rete mucosum especially is hypertrophied, being three to four times thicker than normal; the condition of the horny layer and of the papillæ is extremely variable.

Dermic Papules.—Dermic papules are of two kinds depending on whether the substance which causes the prominence is an edematous fluid or an inflammatory cellular infiltrate.

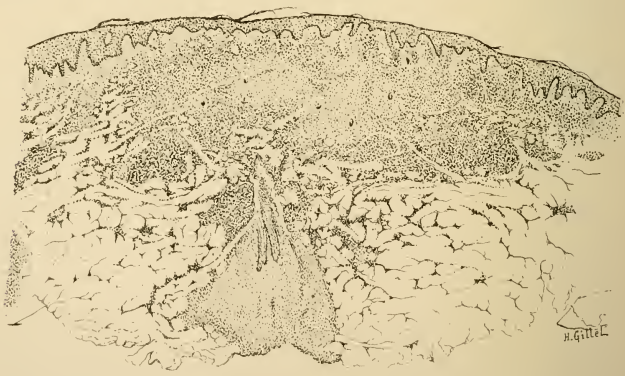


Fig. 31.—Section of a dermic papule. Lenticular papular syphilide. × 25.

The edematous papule is seen in urticaria and papular erythema; the local congestion and exudation of plasma between the meshes of the papillary body disappear in part, at the same time as the blood-pressure, in the cadaver and in excised segments; hardening of the

specimen in alcohol entirely obliterates the lesions. When the erythema-papule is not purely urticarial, but more or less infiltrated, it is found to contain perivascular collections composed mainly of leukocytes.

The most typical example of infiltrated papules is furnished by *lenticular papular syphilides* (Fig. 31). The epidermis here is passively distended, thinned, sometimes with corneal exfoliation. The papillary body and the upper layer of the corium are the seat of a very abundant compact infiltration of cells, among which plasma cells predominate with a few giant cells; at the periphery of the main focus the infiltration consists of perivascular cuffs of plasmocytes.

The papule of lichen scrofulosorum is also essentially dermic; it is found to contain an infiltration of cells of various kinds, lymphoid, epithelioid and giant cells, often arranged as tubercles, either in the papillary body or in the vicinity of pilo-sebaceous follicles.

Mixed Papules.—In the mixed papules there exist combined epidermic and dermic lesions. The papule of lichen planus, which belongs to this type, consists of acanthosis, with more or less keratosis and a limited infiltration of the papillary body; the granular layer is hypertrophied here and there.

The papule of strophulus has a thickened epidermis with localized spongiosis and an edematous infiltrated dermic base.

Clinical Characteristics of the Papules.—A trained observer will have no special difficulty in the clinical distinction of the different varieties of papules. The epidermic hypertrophy manifests itself as a superficial, dry and hard, often yellowish prominence, which is in no way reducible on pressure.

Edematous papules are pinkish-white, tense but compressible; they can be reduced by pressure with the finger-nail, but resume their shape after a few minutes.

The dermic cellular infiltration produces a pink or red papule which is more profoundly indurated, resistant and elastic.

The condition of the horny layer at the surface of the papule is of great importance, depending on the existing dermatosis; it may be thickened or stretched, desquamating in more or less friable and abundant lamellæ or transformed into scales, crusts, and so forth.

The differential *diagnosis* of papules in general is based upon their objective appearance and their course. In the first place, care must be taken not to confuse them with vesicles or pustules, which contain a fluid; with the tubercles of syphilis, leprosy, lupus or with tuberculides, which leave cicatrices; finally, with tumors of small dimensions, which are indefinitely persistent or progressive, such as: various nevi, sebaceous adenomas and hidradenomas, small cysts, circumscribed keratomas, epitheliomas, tumors of molluscum contagiosum, etc.

In some cases a careful examination, pressure under a glass slide (vitropressure), expression of the contents, puncture with a needle or sometimes even the removal and examination of a piece of tissue for biopsy may be necessary.

The following syndromes will be described in this chapter:

Juvenile flat warts, which represent a special clinical variety of common warts. *Lichen planus,* with typical papular eruption, as well as its atypical varieties, which should be studied in this connection, in spite of the irregular features assumed by the eruption; the papules of *prurigo,* as eruptive lesions, the disease as such being discussed in a separate chapter (XXIV); typical *papular syphilides;* finally, the papular form of tuberculides known as *lichen scrofulosorum.*

JUVENILE FLAT WARTS.

The warts designated under this name appear as an eruption of small epidermic papules, not more than 3 mm. in diameter, flattened and barely protuberant; they have rounded or irregularly polygonal contours, are sharply circumscribed, have the color of the normal skin or show a yellowish, grayish or brownish tint; their surface is finely puckered or slightly scaly; they cause no itching.

The warts are met with especially on the face, more particularly the cheeks, temples, forehead and chin, from a few up to several hundred in number. They may also occupy the back of the hands, associated or not with common warts, but are less frequent on the forearms. I have counted over 1500 on a young girl whose face, neck and even chest were dotted with them.

These warts undoubtedly result from auto-inoculation or through transmission from other flat warts, probably also from common warts existing on the patient himself or in persons of his environment. Children, young girls and young women are particularly susceptible. In men, they may be spread by means of the razor.

After having multiplied and persisted for months or sometimes years, these flat warts finally disappear spontaneously without leaving a trace.

The *treatment* must therefore aim especially at avoiding production of scars. Caustic agents must not be used. Painting with an exfoliating mixture is usually prescribed (*Therapeutic Notes,* Section 5). Radiotherapy will cure flat warts with astonishing rapidity; a single session, with moderate dosage, is often sufficient. High-frequency sparks are likewise very successful. Jadassohn emphasizes the efficacy of arsenic treatment. Suggestion probably plays a part in certain cures and the same remark is true for common warts.

LICHEN PLANUS.

The term *lichen* was applied by Willan (and since his time) to dermatoses of different character. At the present time, the existence of a lichen "genus" comprising several species is no longer admissible. With the simple use of the term lichen, we now refer to the great and well defined dermatosis—also known as *lichen planus, lichen ruber planus* or *Wilson's lichen*—to be described in the following.

The expressions: lichen simplex, lichen obtusus, lichen corneus, lichen scrofulosorum, will be defined further on.

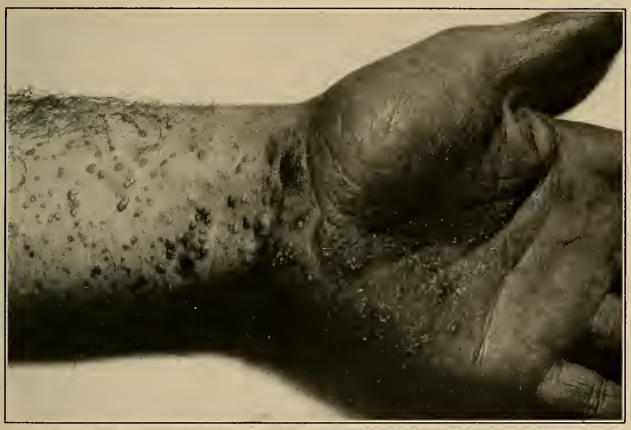


FIG. 32.—Lichen planus of the wrist and the palm of the hand.

Symptoms.—The eruptive lesion of lichen planus is a typical papule, of the average dimensions of a pin-head, of polygonal flattened form, sometimes depressed or umbilicated; its surface is smooth and shining; its consistence is dry and firm; its color varies from a yellowish pink, which is the most common, to a dusky or purplish red; sometimes the color does not differ from that of the normal skin.

These sufficiently characteristic features are often combined with another, which is pathognomonic: the presence of white or grayish opaline streaks and dots marking on the surface of the papules a network or nodular arborizations or stars on a pink background. The "sign of the net," the importance of which was emphasized by L. Wickham, is distinctly visible only on well-developed papules,

which may be isolated or grouped in patches; in order to demonstrate the net, it is advisable to moisten the papules with water, or paraffin oil, or better with anilin oil which renders the horny layers transparent.

The incipient papules are punctiform, pink and glistening; they enlarge in a few days or weeks. Full-grown papules may remain isolated in discrete eruptions, but almost invariably they multiply and become confluent in plaques of very variable extent and round, oval, or irregular shape, usually thicker on the borders than in the middle, of a dusky red or brownish color.

Their surface is covered with fine, very adherent, often scarcely visible scales, but the finger-nail passing over the surface leaves a scaly line. Horny granules are sometimes seen. The papules constituting the patch are sometimes recognizable, especially on the borders. Extensive plaques and patches, when there is complete confluence, are cut in squares by lozenge-shaped or polygonal designs having a smooth and glistening surface, producing a mosaic appearance. On the surrounding skin isolated papules and other groupings may be seen.

Fig. 33.—Lichen planus; patch on the internal aspect of the thigh.

It is the rule for lichen planus, especially when the eruption has existed for a few weeks, to be accompanied by *dyschromia*. The papules and patches become more or less pigmented or become surrounded by a pigmented areola; sometimes, the center is colorless while the circumference is hyperpigmented, brownish or blackish. This symptom may be absent. The coexistence of genuine vitiligo is not very rare.

The most common *localization* of the eruption is the anterior surface of the wrists, the forearms and the legs; but it is also observed on the flanks, the lumbar region, the genital organs, the buccal mucosa, the neck, the palmar and plantar regions, rarely on the face and very exceptionally on the hairy scalp. The eruption may be almost universal.

Pruritus may be altogether absent, often it is slight and intermittent; sometimes, very severe and even excessive to the point of interfering with rest. It may therefore be said that patients suffering from lichen planus scratch themselves, "a little, a great deal, violently, or not at all."

Scratching is undoubtedly responsible for the diffuse lichenization which frequently accompanies the typical eruption, sometimes concealing it.

Varieties.—There exist several varieties of lichen planus characterized by a particular form of the eruptive lesions, or of their arrangement, by certain localizations, or by an abnormal course of the disease.

The papules of lichen planus may assume the form of very regular rings, from 6 to 8 mm. in diameter, with a pigmented center; this form is encountered especially on the genital regions, on the internal surface of the arms, on the flanks and near the articular folds. The existence of a few rings in a typical eruption is not uncommon; they may predominate in a given region, constituting *lichen annulatus.*

Sometimes, in the same regions, the papules are grouped in circles, with centrifugal extension, in gyrate or arabesque patterns; this condition is described as *lichen marginatus seu serpiginosus.*

There also occurs an arrangement of the papules in linear streaks, apparently referable to streaks from scratching: *lichen striatus;* or following the course of a nerve after the manner of herpes zoster or a linear nevus: *lichen zoniformis.*

In certain cases of lichen planus taking an acute or subacute course, the typical lesions may be associated with a few acuminate papules. It might be conceded that this partial and occasional irregularity is entitled to the special name of *lichen acuminatus.* However, at the International Congress held in Paris in 1889, the foreign authors present admitted that the majority of the cases, which with Kaposi they called *lichen ruber acuminatus,* actually belonged to *pityriasis rubra pilaris,* [described by Devergie and long known in France.]

In the palmar and plantar regions (Fig. 32) the papules of lichen are sometimes horny, resembling vesicles at first sight; this desquamation gives rise to a peculiar cribriform appearance. In other cases, the eruption manifests itself in the form of large irregularly outlined red and scaly spots, so that the diagnosis is far from easy.

Lichen planus of the buccal mucosa is especially noteworthy on account of its frequency and its peculiar appearance. Familiarity with its characteristics may assist in the diagnosis of a doubtful case. Ignorance concerning them leads on the contrary to the unfortunately frequent confusion with leukoplakia or syphilis of

the mouth. This localization of lichen is observed in about one-half [one-third?] of the cases; it may be primary, preceding for a long time the cutaneous eruption. Buccal lichen planus is painless and the patients having it are invariably unaware of its existence. The lesion consists either of opaline porcelain-like spots or of a white network, closely resembling, although on a much larger scale, the net which has been described in the typical papules of the skin.

On the genital organs, notably on the glans and prepuce, umbilicated papules and often circinate or annular forms are observed. Vulvar, urethral and anal lichen planus have also been reported.

The ordinary **course** of lichen planus is sluggish. The eruption appears insidiously, progresses during a few months, then persists a variable time, sometimes for years, without changing. Generally, however, this course is interrupted by subacute attacks, for instance on the occasion of physical or emotional disturbances; the lesions multiply, new regions are invaded, there is a recurrence of itching. Retrogression is slow and imperceptible, the papules and patches usually leave very persistent and suggestive pigmented spots.

The name *acute lichen* is used for a variety which takes a rapid course. In these cases, an extensive eruption suddenly appears over large surfaces of the trunk or the limbs. It consists of a diffuse redness, with swelling of the skin and some desquamation, on which can be made out minute incipient papules, the size of a needle-point; sometimes they can be seen only with a lens and when the skin is stretched. Examination by biopsy shows that even the smallest papules possess the characteristic structure. The eruption may be accompanied by some systemic disturbance. More particularly in acute lichen, although only in exceptional cases, a few ephemeral bullæ (*lichen ruber bullosus*), or sometimes acuminate lesions may be seen. Acute lichen subsides in a month or two or it may pass into the chronic form.

Pathological Anatomy.—The structure of the lichen planus papule is characteristic. The Malpighian body, from being at first considerably hypertrophied (acanthosis) is less markedly so later on, when the corneal layer has thickened at its expense. The granular layer is preserved and even hypertrophied (granulosis), but the keratohyalin is unevenly distributed in different points of the same papule. It gives rise to the pathognomonic network of the white or opaline streaks. The horny layer is thickened, coherent, loaded with fat and formed by normal non-nucleated cells (keratosis); in long-standing papules, nucleated corneal cells may be found, and corneal globes are sometimes seen at the orifice of a few follicles.

The papillæ are not elongated, but broadened into cupolas and are often slanting. The boundary line of the derma and the epidermis is therefore very irregular; it is usually somewhat obliterated

in places (Fig. 34, *G*). Sabouraud (1910) has shown that small serous effusions may be encountered in the basal layer of the epidermis, as well as a few giant cells of epithelial origin, either in the basal layer or much more rarely in the subjacent infiltration.

The papillary body contains a diffuse infiltration composed of small round cells; some of these and sometimes a few Malpighian cells, may have undergone colloid degeneration. The lower limit of this infiltration is always markedly distinct; beyond it, only a few perivascular cuffs are demonstrable.

The papule is accordingly of the mixed, epidermo-dermic type.

I have described strictly analogous lesions in lichen planus of the mucous membranes; and in my opinion, the spots and white network are referable to the abundant newformation of keratohyaline.

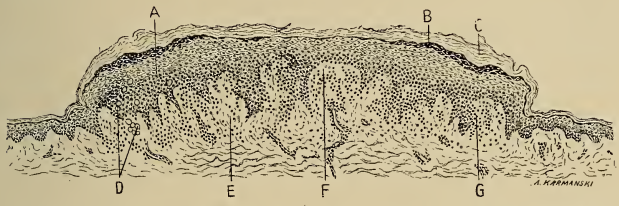


FIG. 34.—Histology of a lichen planus papule. *A*, acanthosis; *B*, granulosis; *C*, keratosis; *D*, colloid degeneration; *E*, diffuse infiltrate; *F*, cupola-shaped papilla; *G*, boundary line of derma and epidermis. × 37.

Diagnosis.—The common error of mistaking lichen for a papular syphilide is not justified by any real resemblance.

Secondary lichenizations have diffuse outlines; glistening facets may be found, but no papules as distinct as those of lichen planus; and the opaline streaks are absent. Real difficulties may arise, however, in some cases.

Pruriginous eczema in children is not infrequently associated with small glistening papules. An observer who is not aware of this possibility may mistake the condition for an eczematized lichen planus or a diffuse prurigo; these papules are generally ephemeral.

Circumscribed prurigo (the chronic lichen simplex of Vidal) occasionally very closely simulates lichen planus. Its papules are hemispherical, instead of plane, less glistening and are devoid of white streaks.

Porokeratosis of *Mibelli*, which is very rare, and the palmar and plantar *keratoderma* and *porokeratosis* may cause serious difficulties in the differential diagnosis from lichen planus of the same regions.

There is a form of *lichen scrofulosorum* with flat and glistening papules, but without an opaline network; it is very rare.

In *lichenoid parapsoriasis,* the analogy of the lesions with those of lichen planus is merely temporary; the course suffices for the differentiation.

Acute lichen may be suggestive of *erythroderma.*

Etiology and Character.—Lichen planus is a disease of adult life, more common in men than in women. [According to the American Dermatological Association's statistics, it is seen once in a little more than 200 cases of skin diseases.]

It occurs so frequently in nervous, irritable individuals, in connection with a psychic shock, violent emotions, grief and worry, accompanied by insomnia, nervous excitement, neuralgias, etc., as to suggest its interpretation as the cutaneous manifestation of a nervous disturbance. This theory cannot be proved. I have not found lichen planus to manifest itself with special frequency during the war. [The nervous disturbances may well be the effect rather than the cause of this dermatosis.]

Jacquet and others have advanced the view that the eruption is always secondary to scratching. It is perfectly true that a patient suffering from lichen planus will sometimes present a linear series of papules originating in a nail or pin scratch. Scratching an affected region may therefore be held responsible for the onset of a more profuse eruption. But on the other hand, itching is often entirely absent and lichen of the mucous membranes is never associated with any pruritic sensation.

The nervous factor is sometimes absent. Apparently contagious cases have been reported, as well as other, more numerous, cases of familial lichen planus.

The pathological anatomy would conform very well with the microbic theory; but this rests on no really conclusive fact. The question of etiology must therefore be left open.

Treatment.—*General Treatment.*—General treatment ranks first in order of importance, being that of nervousness and pruritus. The dietetic regimen, general hygiene, and so forth, must be regulated according to the schedule given elsewhere. (See Therapeutic Notes, Section 12.)

Hydrotherapy—particularly the sedative lukewarm douches advocated by Jacquet as by themselves alone sufficient for the cure of obstinate cases of lichen planus—is often very efficacious. Various hot springs are of value.

Electricity, in the form of static baths and especially high-frequency currents, has yielded remarkable results in my experience. Radiotherapy is applicable to localized eruptions.

Thibierge and Ravaut have observed rapid cures following

spinal puncture and have recommended this procedure in the treatment of the disease; its efficacy is not reliable.

Among internal medicinal agents, *arsenic* is regarded as a specific by the Vienna School and by a number of dermatologists. It is prescribed in various forms and in large doses, but at intervals. Sodium arsenite, for example, may be given in doses of 4 mg. daily to begin with, increasing by 2 mg. daily until 12 to 15 mg. are reached, and then diminishing. Or from 5 to 12 mg. of potassium or sodium arsenite may be administered in daily injections. [Several American observers have reported good results from the internal administration of mercury.]

Under these conditions [when the treatment is prolonged] arsenical poisoning may be expected; aside from digestive disturbances, cramps and formication, there is danger of palmar and plantar hyperkeratosis, or of greatly increased pigmentation of the spots, or even the development of melanoderma. Intravenous injections of arsenobenzol, while not possessing the same disadvantages, cannot be unconditionally recommended.

Antipyrin, nerve tonics, salicylates, etc., are unreliable.

Local Treatment.—Local treatment consists of various topical applications, pastes, salves and plasters made with calomel, yellow oxide, chrysarobin, even with sublimate; to the mercurials may be added phenol, menthol, tartaric or salicylic acids. Collodium with oil of cade is sometimes very successful in discrete eruptions. Applications of potassium permanganate, in fairly strong solution, have been recommended by Hallopeau, even against buccal lichen planus, the treatment of which is extremely unsatisfactory.

Atypical Forms of Lichen Planus.—Of the following dermatoses, some are varieties of Wilson's lichen, whereas others are probably independent of the same and will ultimately come to be classified in other nosographical groups.

Atrophic or **Sclerotic Lichen Planus**, studied by Hallopeau and myself in 1887, is a legitimate lichen planus, the plane papules of which become depressed in the center which becomes cicatricial; they extend slowly at the periphery and become confluent with the neighboring lesions. The resulting atrophic spots are white or nacreous, rounded or polycyclical. On their thinned epidermal lining corneal granules are sometimes found lying in the sweat and follicular orifices. These white spots may attain the size of a silver dollar.

Histology shows that a sclerotic band has become interposed between the epidermis and the infiltration. When the process is checked, the papular border and the rose-colored areola disappear but the cicatrix is indelible (Fig. 35).

The wrists, forearms, neck, breasts, abdomen and sometimes the

thighs are the regions in which this variety of lichen has been especially observed. Zumbusch has described it under the new name of *lichen albus*, which was not needed.

Fig. 35.—Lichen planus atrophicus on the nape of the neck in a woman aged fifty-three years.

Fig. 36.—Lichen obtusus vulgaris of the leg.

Lichen Nitidus of Pinkus is an eruption of small, flat, glistening, sometimes slightly dusky papules, which never becomes confluent and causes no itching. It is almost invariably localized on the penis, but may be generalized, persisting unchanged for years. Histo-

logical sections show a tuberculoid nodule, rich in giant cells, lying directly under the epidermis.

Lichen Obtusus.—The name of lichen obtusus has been applied to various imperfectly understood eruptions with hemispherical papules. *Lichen planus obtusus* of Unna is characterized by dry scattered elevations, the size of a pea, brownish or purplish in color, not scaly, and only slightly pruritic.

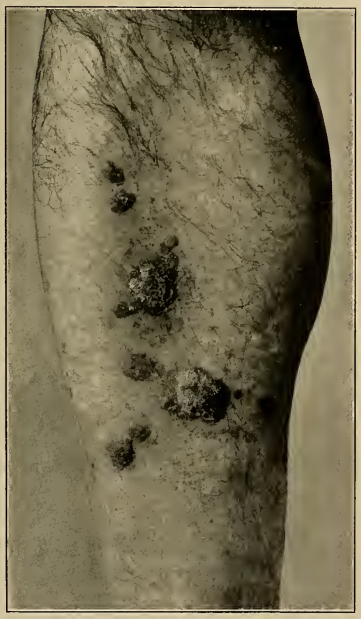


Fig. 37.—Lichen corneus hypertrophicus of the leg, in a man aged sixty-two years, who simultaneously presented lichen planus on the back of the hands.

Lichen Planus Moniliformis of Kaposi seems to be a rare variety of lichen planus, with large hemispherical papules, arranged in strands [like a string of beads].

Lichen Obtusus Vulgaris consists of large, faintly pinkish or brownish papules, usually grouped in a single region and even confluent; I have observed it especially on the anterior surface of the legs (Fig. 36). It is more or less pruritic and runs a very protracted course. The elevations are often covered with a horny coat.

The relations between this form and lichen planus are doubtful; it is often confused with the following:

Hypertrophic Lichen Corneus or Lichen Verrucosus, consists of pinkish or red warty protuberances, usually covered with very adherent brownish or chalky horny masses. They have the dimensions of a pea to those of nummular patches. They are disseminated, or more frequently grouped, or even confluent, in a roughly outlined network. Their surface may have an alveolar appearance, produced by numerous corneal cones dipping into the cutaneous pores. Itching is variable, intermittent and inclined to be nocturnal.

The seat of predilection of the eruption is on the legs, but it may also occupy the elbows, the flanks and buttocks, etc. The microscope reveals a well-marked total epidermic hypertrophy and a considerable elongation of the papillæ.

I have repeatedly demonstrated the coincidence of this lichen verrucosus corneus with typical lichen planus, or with buccal lichen planus, I have also seen lichen corneus developing on oozing or crusted eczematous foci (Fig. 37).

It is, therefore, probable that various dermatoses may terminate in the clinical picture of lichen corneus hypertrophicus, eczemas, prurigos and lichens appearing among these affections. The course is very slow and the disease is extremely obstinate.

The *treatment* of the atrophic and obtuse lichens does not differ from that of lichen planus.

Hypertrophic Horny Lichen requires vigorous washing with soap, applications of caoutchouc or moist dressings for cleansing purposes, followed by strong reducing agents, notably chrysarobin. It is sometimes necessary to resort to the curette or the thermocautery. Hydrotherapy and radiotherapy are very valuable measures in these cases.

PAPULES OF PRURIGO.

Prurigo represents one of the most confused problems in dermatology. The simplest way of settling it consists in calling *prurigo* all primary itching which is accompanied by certain special cutaneous reactions, namely the papules of prurigo and lichenization.

Pruritus and prurigo, as disease conditions, will be described further on (XXIV).

Among their eruptive manifestations, those which assume the form of papules are the only ones here to be discussed; it is desirable to consider them in connection with lichen papules, with which they are always liable to be confused.

There exist two forms of papules in the prurigos: the papule of strophulus or acute prurigo and the papule of genuine or chronic prurigo.

The Papule of Strophulus.—The papule of strophulus is an elevation of the size of a large pin-head, lenticular shape, dusky or pinkish color and firm consistence (Fig. 148). Careful examination shows it to be always centered by a yellowish point which is really a minute vesicle or tiny crust. It originates almost invariably in the center of a more or less transitory urticarial spot. During the first hours, it is often necessary to stretch the skin at the site of this spot in order to bring out the papule hidden by it. The papule will appear like a droplet of wax; on palpation a firm round induration is felt.

At the end of four to twelve hours, the urticarial spot disappears and the papule remains behind. It persists from eight to fifteen days; the crust which crowns it is demonstrable nearly to the end. When this crust has been torn away by scratching, it becomes replaced by a bloody crust. In disappearing, the papule often leaves a not very persistent pigmented spot behind it.

In exceptional cases, smaller papules are met with, lasting only three to four days; larger reddish papules, the size of a lentil; papulo-vesicles with distinct vesicles, sometimes attaining the size of a pea, umbilicated or not, with clear or turbid contents.

Histological examination shows that the strophulus papule is dermo-epidermic and consists of papillary edema and vascular dilatation with a diffuse infiltration of leukocytes; edema of the mucous body; a lenticular disk of colloid appearance situated directly under the horny layer and composed of parakeratotic corneal cells and edematous desiccated epidermic cells; spongiosis is regularly present beneath and on the borders of this disk. This structure is characteristic.

The papule of strophulus, with its associated urticaria, is the special eruptive lesion of acute prurigo simplex, to which its old name of *strophulus* is advantageously reapplied. It is also met with rather frequently, but not invariably, in the first stage of Hebra's prurigo.

The Papule of Prurigo.—The papule of prurigo has the following characteristics: Its volume usually varies between that of a millet seed and that of a large pea; its form is more or less hemispherical, rarely flat; its rounded, sometimes ovaloid contours are not quite distinctly outlined; its color is variable, sometimes of the same shade as the normal skin of the region, or it may have a more or less bright pink, darkened, yellowish or brownish hue; its consistence is more or less firm, never soft; its surface is either very smooth, almost glistening, or more often scaly, not infrequently excoriated and covered with a bloody crust. Briefly, it presents a considerable objective resemblance to the papule of lichen obtusus (Fig. 36).

According to various authors, this papule may sometimes become

crowned with an eczematoid vesicle, or it may undergo suppuration; I believe these are superadded lesions due to traumatism and infection.

The structure of the prurigo papule can be defined in a few words; it consists of localized acanthosis; edema and infiltration are absent or slightly marked; Jadassohn, however, speaks of the frequent presence of large numbers of eosinophile cells in the derma; Leloir and Tavernier have described an intra-malpighian cavity containing clear fluid and few leukocytes, said to be characteristic of Hebra's prurigo; I have never observed it.

The prurigo papule is regularly met with in *Hebra's prurigo* in its second stage, in the form of a scattered eruption; it may reach the size of a hazel-nut in prurigo ferox (see Fig. 149). It is sometimes observed in *diffuse prurigo vulgaris*, but almost invariably in *circumscribed prurigo* (chronic lichen simplex of Vidal). It must not be confused either with the papule of strophulus or with the papule of lichen planus and especially not, as is frequently done, with excoriated follicular papules which will be discussed in describing the other cutaneous manifestations of pruritus and prurigo (p. 487).

PAPULAR SYPHILIDES.

Papular eruptions are among the common manifestations of the secondary stage of syphilis and their recognition is accordingly important.

They are classified, according to the dimensions of the lesions, into syphilides with small papules, which will be described with the folliculoses (for they are always peripilar); syphilides with medium-sized lenticular papules and syphilides with large papules, or papulo-nummular syphilides.

Lenticular papular syphilides are perfectly round, disk-shaped protuberances, of a pinkish (later red or ham-colored) rarely coppery hue, firm to the touch and giving the sensation of a sharply circumscribed dermic infiltration.

On their surface the epidermis is raised in a fine glistening layer; when this is detached, a scaly border is left, known as Biett's collar; the last-named sign may be absent and is moreover not absolutely pathognomonic. Not uncommonly, there is more profuse desquamation and the lesion may then be named *papulo-squamous syphilide*.

Lenticular syphilides are common, sometimes following the roseola through a papular transformation of the spots (*papular roseola*), or becoming intermingled with the latter (*erythemato-papular syphilides*), or they may constitute a syphilitic eruption

by themselves. These attacks have a tendency to recur, especially in the course of the first year, in insufficiently treated patients.

The eruption is usually abundant, symmetrical, very irregularly scattered over the trunk and limbs, even on the face and on the palmar and plantar surfaces. It is often associated with mucous patches, alopecia, sometimes with pigmented syphilides, nodular syphilides or other complicated eruptions.

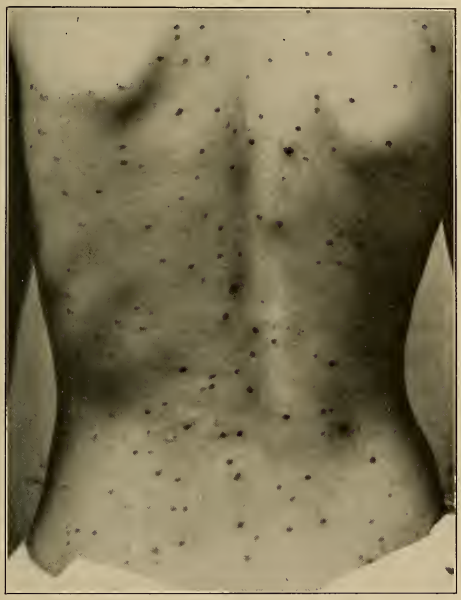


Fig. 38.—Eruption of lenticular papular syphilides, mixed with a few follicular syphilides.

It lasts from ten to thirty days when treated, from two to three months in the absence of specific treatment. The papules leave in their place reddish, sometimes pigmented hyperchromic spots, especially on the lower limbs, very persistent and most distressing to the patients. Such cases were described by Fournier under the name of *syphilides nigricantes*. The occurrence of atrophic spots or macules has also been observed (Fig. 38).

Several deformities or irregularities of the lenticular papules and papulo-squamous syphilides are known to occur. They become

papulo-erosive, under the influence of maceration, in the axillary, inguinal, intergluteal folds, at the umbilicus and around the genitals. This aspect is frequently seen in congenitally syphilitic infants, on the buttocks, the back, the neck, the genital regions and sometimes involves the entire lower limbs.

In the seborrheal regions papular syphilides are arranged in certain cases in mammillate, circinate or irregular-shaped patches, covered with greasy crusts, these are designated as *seborrheal syphilides* and on the forehead constitute the *corona veneris;* they are very resistant to treatment, unless appropriate topical agents are employed together with the specific medication.

The psoriatiform, papulo-crusted and fungoid varieties of the papular syphilides have been mentioned already or will be discussed later.

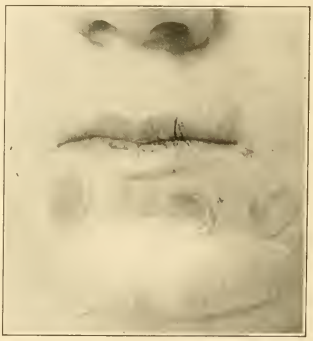


Fig. 39.—Arciform syphilides.

An interesting (because absolutely pathognomonic) form is that of the *papulo-circinate* or *arciform* [or annular] *syphilide* (Fig. 39). This is observed only in the course of the first year and in young women who have already been treated. [It is not uncommon in the negro race in both sexes.] It presents the appearance of regular rings, graceful arcades or complicated circinations, on the chin, around the lips and nostrils, or sometimes on the vulva. The circles are continuous or formed by small fine-scaly papules having a yellowish-pink color.

Papulo-nummular syphilides are discoid or oval prominences, from 1 to 3 cm. in diameter; on account of their form and their ordinarily oozing or crusted surface, they have been named *cutaneous patches* (Bazin), *syphilitic patches* (Legendre), or *mucous patches of the skin.*

These syphilides are extremely common in the ano-genital region, but may develop in any skin fold. They are also met with in association with a papulo-lenticular eruption, on the neck, the face, the shoulders, etc., but are not numerous there. They might be confused with iodide eruptions, but these are pustular and grow much more rapidly.

All these papular syphilides have a nearly identical structure (Fig. 31); they are composed of an intradermic plasma-cell infiltration and differ among themselves only by the lesions of the epidermis.

The *diagnosis* of papular syphilides is usually easy. Lichen planus, psoriasis and parapsoriasis differ from these syphilides by the features of their own lesions. The papules of the diffuse and circumscribed prurigos are distinguished by the very active pruritus by which they are accompanied. Hydradenomas of the thorax, which are very rare, have an analogous appearance but their duration is indefinite and their localization is strictly limited.

Papulo-necrotic tuberculides may give rise to serious diagnostic difficulties, although it is known that they chiefly affect the limbs, that their development is successive and usually slow, that they become hollowed out by ulceration and leave a cicatrix. In a given case, however, the decision may have to be based upon the coexistence of other specific manifestations, biopsy and serodiagnosis. The diagnosis of posterosive syphiloids in the newborn is still more difficult. Mistakes are common and seriously detrimental to infants wrongfully suspected of syphilis. The presence of other manifestations of congenital syphilis, the condition of the parents and the course of the eruption, permit a positive diagnosis in some cases. The Wassermann reaction of the child and its mother must be determined in doubtful cases and sometimes biopsy and a search for spirochetes is called for.

LICHEN SCROFULOSORUM.

Lichen scrofulosorum is a papular eruption resembling lichen in its clinical appearance, but in its nature related to the tuberculides or attenuated tuberculoses of the skin.

It will be seen further on that the tuberculides constitute a group of dermatoses of identical etiology, but extremely polymorphous objective characters. The most frequent and distinct forms have received special names, the others are considered as intermediary forms.

My description deals with the most pronounced type of the papular tuberculides; but it must be emphasized from the start

that there exist numerous atypical and less distinctly characterized varieties.

Lichen scrofulosum of Hebra, the papular scrofulide of Bazin—a better name for which would be *lichenoid tuberculide*—presents the following appearance:

An eruption of small papules, of the average size of a pin-head slightly prominent, flattened, polygonal, of a pale yellow or more rarely a dusky red color, of rather soft consistence, a surface smooth and glistening or more commonly covered by a slightly adherent scaly layer, such is the typical form (resembling the papule of lichen planus) which is, however, not the most frequent.

These papules are almost invariably grouped in more or less numerous nummular plaques or in patches, rings, or semicircles; also as an irregular network, the interpapular areas remaining normal.

The eruptions usually occur on the trunk, especially the flanks and loins, but may spread to the limbs and exceptionally to the face. It appears insidiously, almost without pruritus, persists during several months and then disappears leaving no trace of any kind. Sometimes recurrences develop in attacks during several years.

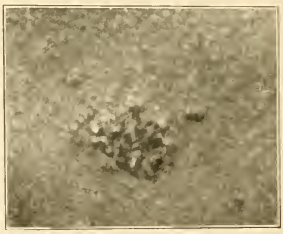


Fig. 40.—Lichen scrofulosorum, acuminate variety. (This group of papules suddenly appeared on the back of a young woman suffering from lupus, shortly after an injection of tuberculin).

There are numerous *varieties*, and the following forms may be found associated with the flat and glistening papules, in the same or in different regions, or constituting by themselves the entire eruption: conical or acuminated papules, of a rose-red color (Fig. 40) and centered over a follicle the hair of which may be broken off flush with the orifice; in other cases, conical papules crowned by a vesico-pustule, of the type of acne cachecticorum; or again, confluent lesions may form scaly polygonal disks, of a purplish or brownish-red color, slightly infiltrated, not very prominent, which resemble psoriasis or some of the eczematides; and finally, the eruption may present a mixture of follicular papules and

patches dotted with acuminate lesions suggestive of pityriasis rubra pilaris or lichen spinulosus.

Under the heading of lichen scrofulosorum there should also be grouped, according to Boeck, as deformed or attenuated varieties: his disseminated *papulo-squamous tuberculide;* his *eczema scrofulosorum,* characterized by crusted and oozing patches with an infiltrated base constantly recurring at the same points; certain forms of *pityriasis simplex* of the face in children, arranged in circumscribed, red, fine-scaling patches and accompanied by large cervical glandular swellings.

Lichen scrofulosorum is observed at all ages, but occurs by predilection in children and youthful individuals suffering from glandular or bony tuberculosis or from sluggish visceral tubercular lesions. It has been known to follow acute diseases, such as measles, etc.

The papules are formed, as was first shown by Jacobi and Sack, by a dermic infiltration having almost invariably the constitution of characteristic tubercular follicles, with giant cells, but with a slight tendency to caseous degeneration; these tubercles are situated in the papillary body or around a pilo-sebaceous follicle.

Jacobi, Sack and Wolff succeeded in finding the Koch bacillus in their specimens and in a few of their cases guinea-pig inoculation proved positive.

A general and local tuberculin reaction is practically constant; Jadassohn obtained a positive reaction in 14 out of 16 cases. Schoeninger, Buzzi and myself have observed the appearance of lichen scrofulosorum immediately after injections of tuberculin, which undoubtedly merely served to bring out latent lesions (Jadassohn, 1896).

The *diagnosis* of lichen scrofulosorum may present very considerable difficulties. In view of its habitual polymorphism, it is advisable to examine not only one eruptive group but the eruption as a whole; thereby guarding against confusion with the eczematides, lichen planus, acne cornea, abortive forms of pityriasis rubra pilaris, etc.

The differential diagnosis from the follicular lichenoid syphilides is sometimes almost impossible, even on examination by biopsy. The Wassermann reaction is of great help in these cases.

The *treatment* is that of tuberculosis in general: fresh air, sunlight, cod-liver oil, calcium compounds and arsenic; tuberculin in very minute doses or novarsenobenzol may lead to rapid improvement. Locally weak reducing agents and cod-liver oil ointments have been recommended.

CHAPTER VIII.

VESICLES AND VESICULAR DERMATOSES.

Vesicles are small circumscribed elevations of the epidermis containing a clear fluid. Their size varies from that of a pin-head to that of a pea; their form is hemispherical, or sometimes acuminate or umbilicated; their outline is round; or, as the result of confluence they may assume an angular or polycyclical configuration.

The contents of the vesicles are fluid and transparent like water or yellowish and serous. It is sometimes necessary to prick the roof with a needle in order to demonstrate the presence of the fluid and its properties. The contents are apt to become cloudy or turbid after a time; sometimes they are hemorrhagic from the start.

As the result of desiccation, the vesicles are transformed into crusts whose origin is indicated by their shape and arrangement.

On the mucosæ and semimucosæ, as well as in regions where skin rests against skin, the vesicles rupture very readily leaving red erosions or often diphtheroid lesions of rounded or polycyclic contours.

Modes of Formation of Vesicles.—They always result from an accumulation of plasma in the epidermis. Vesiculation may occur in three different ways which are, however, frequently combined.

In the first, or *parenchymatous vesiculation*, the fluid accumulates first in the interior of the Malpighian cells and the resulting unicellular vesicles become confluent with each other. This so-called "altération cavitaire" of Leloir predominates in smallpox, vaccinia, etc.

In a second type, or *interstitial vesiculation*, the edema is intercellular, compressing and stretching the Malpighian cells which are drawn out into a net, the threads of which finally rupture, producing the spongioid state of Unna, the spongiosis of Besnier. The vesicles of eczema are produced in this way (Fig. 5).

In a third type, the edema is likewise intercellular, but the cells become globular, are detached from each other and float in the fluid, undergoing a cloudy or fibrinous degeneration, sometimes becoming hypertrophied with multiplication of their nuclei. This is the *ballooning alteration* of Unna, which predominates in the vesicles of varicella, zona (Fig. 41), herpes, etc.

Whatever the mechanism of their production, vesicles always

result from an inflammatory process and constitute acute eruptions on a hyperemic base.

Vesicles differ from *bullæ* not only by their usually smaller volume but also by their mode of formation. In contradistinction to bullæ, they are frequently multilocular, at least at their inception.

They differ from serous cysts, notable from the hydrocystomas and lymphatic varices which may simulate them, by the intra-epidermic seat of their fluid; they can be opened by puncture without causing hemorrhage.

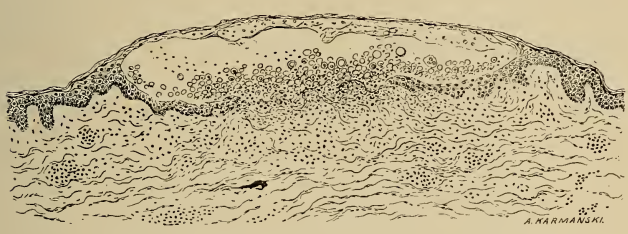


FIG. 41.—Section of a zona-vesicle on the tenth day. Vesiculation through ballooning alteration; in the center of the vesicle all the cells of the rete are degenerated and the papillæ are exposed. In the cutis an abundant infiltration of lymphoid cells is seen. × 50.

Vesicular Dermatoses.—Vesiculation is observed in the eruptive fevers, variola, varicella and vaccinia, for which the reader is referred to text-books of general medicine.

In eczema from any cause, vesiculation is very common; it has even been considered as characteristic of the eczematous process. It may be absent, however, and at any rate represents merely a stage or episode of *eczema*, which was discussed in a chapter by itself (IV).

The vesicles of cutaneous trichophytosis, formerly called *herpes circinatus* seem to me to be of eczematous character; they will be mentioned in the chapter on parasitic dermatoses, as will also those which complicate scabies.

Sudamina, notwithstanding their small size, are not vesicles but minute subcorneal bullæ; they will be discussed together with the hidroses.

In recurrent polymorphous pemphigus, commonly known as *dermatitis herpetiformis* of Duhring, genuine vesicles are found at the same time as real bullæ; this affection is in every respect related to the bullous dermatoses.

The eruption of *strophulus* is usually papular; it is exceptional for the microscopical vesicle which crowns its papules to assume a

development rendering it visible to the naked eye; however, certain very rare cases have simulated varicella. This affection belongs to the group of the prurigos.

In this chapter there remains to be studied only herpes, zona and the zosteriform eruptions. These are purely and exclusively vesicular eruptive syndromes.

HERPES.

Herpes is an acute eruption of a cluster of vesicles, varying in number and originating on an erythematous base, situated anywhere, although having a predilection for the face, around the mouth and the nose and for the genital region. The affection is extremely common and known to the laity under the name of "fever blister."

The word herpes formerly had a much more restricted significance. The terms *herpetism, herpetides,* no longer possess any accurate meaning. The name of herpes is still used for some dermatoses of very variable kinds, such as herpes circinatus, herpes gestationis, herpes iris, herpes cretaceus, etc., which are in no way related to genuine herpes. Zona is also known as herpes zoster.

Symptoms.—Frequently preceded a few hours by shooting pains or a tense and burning sensation, herpes appears in the form of a congestive edematous spot, which promptly becomes covered with uniform vesicles, the size of a pin-head, with clear contents, from two or three to several dozen in number. These vesicles lie very close together and sometimes become confluent. They are rarely hemorrhagic. There may be several as if accidentally scattered groups. The lymph glands are slightly enlarged.

The herpetic vesicles became turbid, then opaque and shrivel up, forming yellow or brown crusts which become detached at the end of eight to ten days, leaving a temporarily red spot, but never a permanent scar. Herpes has a marked tendency to recurrence.

The seat of election of herpes is the lips, the nostrils, or any part of the face; or the genital regions. Herpes is also not infrequently seen on the lobes of the ears, the nipples and the mucous membranes; but is much rarer on the trunk and limbs. As a rule, only one of these regions is affected at the same time. In the course of the War, however, I was enabled to observe in young soldiers recently vaccinated against typhoid fever, profuse eruptions of herpes occupying simultaneously the forehead, the nose, both lips, the mouth, the chin, the ears, the neck, and in one instance one of the upper extremities; the condition being suggestive of so-called multiple zona.

Herpes genitalis [or *progenitalis*] is especially noteworthy on account of the liability to misinterpretation. *In men,* it occupies the balano-preputial sulcus, the glans, the prepuce, or more rarely

the meatus. On the parts covered by the clothing, it is rapidly transformed into erosions. These are very superficial, isolated or confluent, round or polycyclic and microcyclic, red or diphtheroid; some fluid can be squeezed out of them and they are slightly painful. Herpes is not indurated at the base, provided it is not improperly treated or abused, and heals in at most eight or ten days. [On the glans a certain degree of induration is common and must be considered in making a diagnosis.] When the lesion has been cauterized or treated with irritants or antiseptics (silver nitrate, sublimate, tincture of iodin, aristol, etc.), always an improper procedure, it may become indurated, associated with inflammatory phimosis and painful swollen glands, ulcerate and last several weeks. In such cases an immediate diagnosis is extremely difficult, sometimes impossible before the inflammation has been relieved by soothing applications.

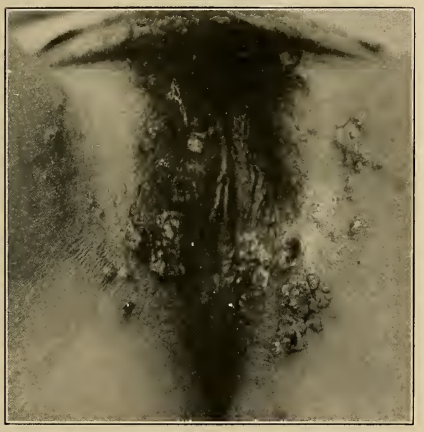


FIG. 42.—Profuse vulvar herpes, showing eruption of relatively moderate severity.

In women, herpes may occupy any portion of the vulva, presenting the same features as in the male. It is met with exceptionally in the vagina and the cervix uteri.

A profuse vulvar herpes sometimes occurs (Fig. 42) with some fever, severe burning sensation, very marked local edema, extensive patches covered with agglomerated or confluent vesicles, extending from the vulva to the pubis, the internal surface of the thighs and in the intergluteal fold. The macerated vesicles rapidly break, are transformed into erosions or become covered with a diphtheroid layer and secrete an offensive muco-purulent discharge. The glands

are swollen and painful. The patient suffers great inconvenience and is confined to bed. Healing takes place in fifteen or twenty days. In some cases under my observation, the lesions became prominent after epidermization had occurred, closely simulating papular mucous patches.

Aside from the inconvenience and actual pain caused by it, genital herpes is important on account of its tendency to recurrence and because it prepares the soil for severe infections, soft chancres and syphilis, in persons exposing themselves to risk of these diseases.

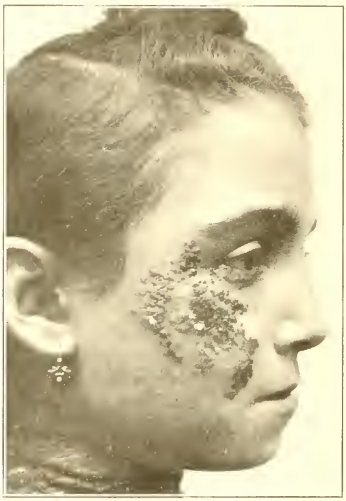


FIG. 43.—Recurrent herpes of the cheek. A photograph of the same young girl, taken two years previously by Brocq, is shown in his Traité de Dermatologie, ii, p. 280; illustrating a more discrete eruption in the same area.

It must also be kept in mind that an eruption of herpes may precede or accompany the onset of a syphilitic chancre which may consequently pass unobserved or remain unrecognized for some length of time.

Herpes buccalis is less common, and is usually met with in connection with a profuse herpes of the lips or the face; it may occupy the mucosa of the cheeks, the palate and the tongue. It is often bilateral. Its vesicles are ephemeral.

Herpes of the pharynx seems to constitute one of the varieties of *herpetic angina*, characterized by the abruptness of its onset, the

rapidly rising temperature, the severity of the local pain and the marked general phenomena. Vesicles are occasionally found, but more often polycyclic erosions, diphtheroid or not, which succeed them. In doubtful cases, these erosions can be brought out, as on all mucous membranes, by painting them with a weak solution of silver nitrate, or better with a watery solution of chromic acid, 1 to 50 (L. Jullien). The glands are usually swollen and painful.

Herpes conjunctivalis represents one of the forms of phlyctenular conjunctivitis.

Recurrent herpes is that form which recurs, not accidentally and at any point, but with a certain periodicity and in a practically constant region.

Certain women have an eruption, either around the mouth or on the genitals, at nearly every menstrual period (*catamenial herpes*).

Recurrent progenital herpes, more particularly observed in men, is considered by Diday and Doyon as related to an existing or preceding venereal disease, especially soft chancre, gonorrhea and syphilis.

Recurrent herpes of the face affects children and youthful individuals, returning periodically several times in the course of the year on the same cheek, for ten years or more (Fig. 43).

Recurrent herpes of the buttock occurs in adults of both sexes, and takes a more variable course.

Recurrent herpes of the mouth, pointed out by A. Fournier, the distress of some old syphilitics, is referred either to remains of long-cured specific lesions, or to abuse of mercury.

These various recurrent herpetes are not infrequently associated with *neuralgic* pains, burning sensations, painful tension, glossodynia, etc.

Etiology.—Herpes is observed at any age, but especially during youth and maturity. Its causes are complex. There undoubtedly exists a traumatic herpes; dentists, for example, are familiar with the herpes developing around the mouth as a sequel of dental operations; on the other hand, herpes of the vulva is not uncommon after the first sexual intercourse. In both cases, the patients are much inclined to suspect that they have been infected.

Many cases of recurrent herpes of the nose, the lips, etc., have seemed to me to be related to a chronic irritation of the neighboring cavities, dental caries, alveolitis, pulpitis, gingivitis, rhinopharyngitis, sinus inflammations, otitis, etc. The nervous and probably *reflex* character of many cases of herpes is therefore obvious. On the other hand, there are cases of symptomatic herpes, in certain infections. Herpes is known to be common in pneumonia, epidemic cerebrospinal meningitis, influenza, etc. Sometimes a high fever of a few

days' duration is terminated by an eruption of herpes, whence the assumption of a herpetic fever.

A fairly well marked lymphocyte reaction of the cerebrospinal fluid was noted by Ravaut and Darré in 21 of 26 cases of genital herpes.

The disease is not contagious, nor can it be inoculated. Nevertheless, it is difficult to reject the idea that such an acute and severe dermo-epidermic lesion often accompanied by glandular enlargement, constitutes an [infectious] inflammatory reaction rather than a "trophic disturbance."

Treatment.—Herpes of the skin requires no active treatment, it suffices to cover it with a bland powder; herpetic eruptions can sometimes be aborted by dressings with absolute alcohol (90 per cent), or with borated or camphorated alcohol; but failures are the rule. Watery dressings and ointments are injurious, except in the stage of complete desiccation.

Genital herpes must be gently treated; all irritant applications involve the risk of changing its appearance and prolonging its duration. It suffices to wash or bathe it with lukewarm boiled or borax water, or an infusion of bran; to powder it with talcum, or zinc oxide, or bismuth subgallate. Touching with a drop of silver nitrate, 5 per cent., is permitted and useful, but only when the diagnosis is positive and when the erosion fails to become covered with epidermis at the end of a few days. [Inasmuch as a herpes may lodge a spirochetal infection cauterization must in general be avoided.] Profuse herpes of the vulva requires rest in bed; the pain is relieved by poultices of starch or cooling creams.

In the case of recurrent herpes, the essential point is the discovery of the initial focus of chronic irritation and its removal. Radiotherapy has repeatedly proved successful in my experience in the cure of recurrent herpes of the buttock and the cheek.

ZONA.

Zona—or herpes zoster—is an acute eruption of vesicles, grouped upon erythematous patches and ordinarily located along the distribution of a nerve, on only one side of the body. Its development is approximately cyclic; it recurs but rarely.

Symptoms.—The eruption appears suddenly and is accidentally discovered by the patient, or it may be preceded by prodromata and accompanied by pains.

At the onset, nothing is seen but slightly elevated erythematous patches, with a shagreened surface, of oval or irregular outlines, from a single one to twenty in number, generally half a dozen, separated by areas of healthy skin. At the end of a few hours,

at most a day, vesicles form first in the center, then on the entire patch, and rapidly increase in size; they become tense, pearly, uniform, from the size of a small to a large pin-head. They lie close together, more rarely discrete, sometimes confluent.

Their fluid content becomes opalescent and turbid, even purulent, by the third day, while at the same time the plaque fades and flattens; desiccation begins from the fourth to fifth day and is completed by the eighth to twelfth day; the crusts do not fall off until the end of twelve to twenty days.

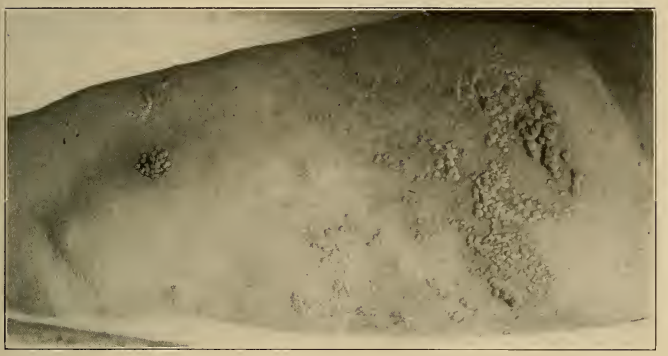


Fig. 44.—Zona, on the tenth day of the eruption, occupying especially the cutaneous territory of the II and III left lumbar roots. Near the groin the patches are confluent, the largest vesicles are hemorrhagic; numerous incomplete vesicles are also seen. Near the knee the patches are isolated and typical.

The onset of the various patches is usually not simultaneous but successive, in the course of two or three days, so that several stages can be observed at the same time (Fig. 44). It frequently happens that the eruption remains incomplete on certain patches, especially the last to appear. From the start, or at the end of one or two days, the fluid of the vesicles may assume a sanguinolent character, constituting *hemorrhagic zona.*

It is rare for the vesicles to rupture. When they are opened in the stage of suppuration, erosions or even fairly deep ulcerations which heal slowly are found beneath them; in this case, the eruption leaves behind it, not only brownish macules, as is usual, but indelible white scars with pigmented halos, sometimes cheloids, having a characteristic distribution. This possibility must be mentioned to the patient.

In cachectic or weak and senile individuals, genuine sloughing

scars may supervene, representing *gangrenous zona*, with a prognosis sometimes serious.

The lymph glands corresponding to the territory of a zona are nearly always enlarged, sometimes before the eruption appears. Outside of this territory, the skin is healthy. However, the possible presence of *aberrant vesicles*, at a distance from the regional eruption, has been pointed out by Temeson. In a contribution to this subject (dedicated by me to Professor Barduzzi, of Livorno, in 1911) it was noted that this observation is correct, but that, leaving out of consideration the ordinary follicular inflammations and lesions of miliary impetigo which may accompany the disease, genuine aberrant vesicles in zona are relatively rare. [During an outbreak of smallpox in New York in 1900 I observed several cases of generalized herpes zoster in which the eruption was so extensive as to require careful examination before varicella could be excluded.]

Pain is variable or may be absent. Almost invariably, however, zona is either *preceded* for several days or even several weeks, or *accompanied*, which is more common—or *followed*, by *neuralgic pains;* these pains are continuous or paroxysmal and may assume all possible forms, especially a severe burning sensation (hence the popular name of St. Anthony's fire). The patches themselves are sometimes anesthetic, it is claimed, but really almost invariably hyperesthetic. The frequency, severity and duration of the pains are dependent upon the age of the patient; they are usually absent in children, but in aged individuals may persist indefinitely, causing extreme distress.

The eruption of zona may be preceded by *general phenomena*, malaise, prostration, anorexia, with a transitory fever of 39° or 40° [102°–104°]. In this case, the infectious behavior of the disease, its sometimes epidemic and immunizing character, have caused it to be likened to the eruptive fevers: the *zoster fever* of Landouzy and Erb. The course of the disease may be described as nearly cyclical.

Among the *localizations* of *zona*, the most common is *intercostal zona*, to which the disease owes its name. It covers the thorax or the abdomen like a half-belt, without passing more than a few millimeters at most beyond the median line. When it occupies the territory of the first intercostal nerves, it follows the anastomotic branch which the second intercostal nerve sends to the internal brachial cutaneous nerve down the inner surface of the arm.

Cervical zona, which affects all or only some of the branches of superficial cervical plexus, is likewise fairly common and the same remark applies to *lumbo-abdominal* and *genito-crural zona*. *Peripheral zonas*, affecting the limbs, are more unusual.

Ophthalmic zona, corresponding to the superior branch of the trigeminal nerve, is frequent and grave on account of its possible

sequelæ. It presents frontal, palpebral, nasal, even pituitary patches and in two-thirds of the cases gives rise to ocular lesions, especially of the conjunctiva and cornea, lesions of the iris and amblyopia being less common. Anesthesia of the cornea, corneal perforation, irido-choroiditis and retinitis have been reported but are very rare. Ophthalmic zona must not be confused at the start with erysipelas or with kerato-conjunctivitis from other causes.

Zonas of the mucous membranes, buccal, pharyngeal, etc., are of very exceptional occurrence.

Zona is almost invariably unilateral and may occupy the territory of a single nerve; as a rule, however, it spreads over the territory of two or even three neighboring spinal roots,

Exceptionally, cases of *double* or *bilateral, alternating, multiple,* or even *generalized* zona are encountered, such as a case reported by Colombini in 1893.

Zona usually occurs only once in a life-time, but recurrences have been noted in a few instances. So-called zonas with multiple recurrences belong to the zosteriform eruptions.

Etiology and Pathogenesis.—Zona affects all ages and both sexes equally. It is slightly more common in the spring. The cause as a rule remains entirely obscure. It has been observed to follow after severe traumatisms, violent emotions; to occur in the course of infections or general diseases, at the beginning or in the course of tuberculosis (Leudet), pneumonia, pleurisy, syphilis, cerebrospinal meningitis, mumps, carbon monoxid poisoning, arsenic medication (Hutchinson, Neilson), diabetes, cancer, etc.

A number of circumstances indicate the *infectious nature* of zona, at any rate in many cases. It is true that the disease is not contagious, but it sometimes seems to be vaguely epidemic, in the sense that several persons from the same environment are attacked at intervals of a few days. It may be accompanied by general phenomena; glandular swelling is usually present. Sabrazès and Mathis have demonstrated the occurrence of hyperleukocytosis in the course of zona, especially of the polynuclears and the eosinophiles. Finally, an attack of zona usually confers immunity against another. The pathogenic agent, however, has not as yet been demonstrated.

On the other hand, the relations of zona with the *nervous system* are very striking, in the distribution of the eruption as well as the associated neuralgias. I have seen a case of ophthalmic zona following upon a severe contusion of the skull in an automobile accident. Mme. Dioudonnat-Lempert devoted her thesis (1914) to the study of cephalic or cervical zona of dental origin. Lesion of a nerve branch in the course of a mercurial injection may give rise to a cluster of zona-vesicles along its area of distribution. Otic or para-auricular zona is not infrequently associated with facial paralysis.

It has recently been shown that in the course of syphilis, zona may indicate a premature syphilitic meningitis which affects the posterior roots, or it may precede tabes and general paralysis. Zona is also sometimes encountered in syringomyelia, in dementia, vertebral tuberculosis, cancer of the vertebra, etc. It is often accompanied by lymphocytosis of the cerebrospinal fluid; in different cases, this may exist prior to the eruption, coincide with it, or follow several days later; or it may be entirely absent. More rarely, rigidity at the nape of the neck, Kernig's sign, retardation of the pulse, headache, etc., have been noted.

Numerous authors have demonstrated neuritis or degeneration of the *nerves* in the affected area. However, as the distribution of zona is far from being always in exact conformity with the distribution of the corresponding peripheral nerves, a lesion of the spinal root and especially of the spinal ganglia is now held to be responsible. Following Baerensprung and Charcot, various observers have formed hemorrhagic, inflammatory, or degenerative lesions of the spinal *ganglia* and the posterior roots in cases of zona; among these writers, Head and Campbell must be specially mentioned (in 31 cases). Head in England, Brissaud, Achard, and others in France, have endeavored to refer zona to a lesion of a spinal or metameric segment.

At the present writing, the conclusion seems justified that the *metameric theory* is probably true for certain cases; that the *radicular and ganglionic theory* serves to explain the great majority of zonas, as has been confirmed by anatomical findings; finally, that the *peripheral nervous theory* applies to certain rare cases.

Granted that the necessary pathogenic condition of zona is a lesion at some point in the course of neurons which supply the affected territory, one is justified in admitting that this lesion can be, in different cases: (1) of traumatic origin; (2) of toxic origin (arsenic, CO); (3) of autotoxic origin (diabetes); (4) finally, of infectious character, either ordinary (tuberculosis, syphilis, pneumonia, grippe, etc.), or sometimes due to a specific germ. The probable existence of this special microbic agent, which still remains unknown, would serve to explain the epidemic character, the prodromata, the febrile and infectious symptoms, the aberrant or disseminated vesicles, the immunizing power against a new attack which is characteristic of certain zonas and the so-called zoster-fever of Landouzy and Erb, or *idiopathic zona*.

Jadassohn points out that according to this interpretation, zona is the perfect type of a *syndrome* with an always identical morphology and pathogenesis, but a variable etiology.

Diagnosis.—In typical cases, this is extremely easy; the eruption of clustered vesicles, arranged in groups over one or more nerve territories, unilateral, accompanied by pain and taking a cyclic

course, is absolutely characteristic. Doubt is possible only in incomplete or abortive cases. A careful examination will guard against confusion with *erysipelas, eczema,* or *polymorphous erythema.* The differential diagnosis may occasionally be difficult from herpes and the zosteriform eruptions.

Herpes is identical as regards the eruptive lesions, but is sufficiently characterized by its site, its frequently bilateral appearance, and its liability to recurrence. In the very unusual case of a single patch of zona, or in the presence of certain bilateral zonas, the differentiation may prove practically impossible.

The *zosteriform eruptions* constitute a somewhat indefinite group, including rare and rather dissimilar observations. This name is applied to exanthems constituted by less numerous, less definitely grouped and more voluminous lesions than those of zona, occupying the territory of one or several nerve trunks, but only on a limited portion of this territory, accompanied by sensory disturbances, muscular atrophy, vasomotor disturbances, etc. Their essential characteristic is their repeated recurrence during several years. In one of my patients, vesicles reappeared five or six times a year at different points of the left hand, but always on the cutaneous territory of the median nerve, throughout a period of ten years.

Zosteriform eruptions, sometimes called *chronic zona* or *recurrent zona,* are observed after traumatism of the nerves, in peripheral neuritis, in diseases of the cerebrospinal axis, in syphilis, malaria, etc.

Certain *recurrent zonas* might be classified equally well among the zosteriform eruptions, but the eruption is here far from being invariably unilateral.

Treatment.—The local treatment of zona must be as simple as possible; avoiding moist dressings, poultices, salves, and ointments, which only serve to favor infection, leading to ulceration and scar-formation. It is advisable to empty the largest or confluent vesicles by pricking them with a flamed needle and to apply large amounts of bland, aseptic or sterilized powders, in order to favor desiccation. Occlusive dressings will relieve the painful sensations.

Ulcerative or gangrenous lesions are treated in the usual way. The sometimes extremely distressing pains of zona require special treatment. Analgesic agents, aconite, gelsemium, antipyrin, pyramidon, exalgin, aspirin, are sometimes sufficient. Morphin injections must be avoided, if possible, on account of the danger of the morphin habit. Injections of a cocain solution, or sterilized air, along the course of the affected nerve, have been useful in a few cases. Radiotherapy and galvanic electricity are valuable measures in cases otherwise regarded as desperate.

For the treatment of ophthalmic zona, the reader is referred to text-books on ophthalmology.

CHAPTER IX.

PUSTULES AND PUSTULAR DERMATOSES.

The eruptive lesion known as a *pustule* is an epidermal elevation containing a purulent fluid. The cavity containing the pus may be situated in the epidermis, in the cutis, or in a follicle. A distinction is accordingly made between:

1. *Epidermic pustules* which may be *superficial* when formed under the horny layer (example: impetigo); or *deep*, when they involve the basal layer of the mucous body and therefore leave a scar behind them (example: variola, ecthyma); (2) *Dermic pustules*, which are rare (example: miliary abscesses of the newborn; pustules of tuberculosis verrucosa); (3) *Follicular pustules*, which on the contrary are extremely common. The two last-named varieties will be discussed in other chapters.

It would be correct and logical to reserve the name *pustules* for primary pustules, namely, those in which the lesion is suppurative from the start. In case of secondary suppuration of other eruptive lesions, terms should be employed like *suppurative vesicles, purulent bullæ*, or *suppurative papules*, according to the condition present; but as this distinction is not always practicable; the words *vesico-pustules, papulo-pustules*, and *tuberculo-pustules*, are in common use to obviate this difficulty.

Collections of pus in the hypoderm are not pustules, but *abscesses* or *gummas*.

Pustules are of rounded, rarely oval configuration, more or less prominent, hemispherical or flattened, tense or flaccid, of a yellowish white or grayish color and surrounded by an inflammatory areola.

Their dimensions are extremely variable, they may be punctiform, lenticular, or nummular; often they are small at first and undergo a centrifugal enlargement.

It is usually easy to ascertain their more or less deep seat by means of direct examinations; if necessary—and it is advisable to do so—they must be punctured with a needle, in order to empty the contents, determine the thickness and constitution of the roof, the characteristics of the floor of the pustules, etc.

The contents may be a more or less turbid and yellowish fluid, or consist of the creamy thickened substance known in former times as "laudable pus." Examined under the microscope, it contains chiefly polynuclears and plasma.

The pustules do not persist a long time, they terminate by accidental or spontaneous rupture, or by desiccation. In both cases, they are followed by a yellow, brown or black crust, more or less thickened and irregular, covering an erosion, excoriation or ulceration.

The *crusts*—concerning which a few words are here in order from a general point of view—are concretions resulting from the desiccation of serous fluid, pus, or blood. Their thickness, regularity, more or less hard, greasy or friable consistence, their color ranging from light yellow to deep black and their adherence vary in extremely wide limits and are suggestive of their origin.

Crusts form on wounds, erosions, traumatic or pathological excoriations of all kinds, on ulcerations and on old vesicles or pustules. In case of vesicles or pustules, the epidermis regenerates itself underneath the lesion, extending from its periphery toward its center, eliminating it in the form of a crust; this mechanism is designated as eviction.

Crusts are absent from tissue surfaces kept moist by mutual contact or by dressings.

An essential difference exists between crusts made up of *scales* which are disintegrating epidermal layers; and *hyperkeratoses*, in which coherent horny collections are formed.

A concretion is sometimes formed by epidermal layers alternating with layers of dried serum or pus; these *scaly crusts* have a leaf-like structure and oily consistence; they are observed in a variety of conditions, notably in the eczematides.

Pustular Dermatoses.—The dermatoses in which primary or secondary pustules occur are extremely numerous.

1. In the first place the *pyodermatitides* are characterized by pustules from the start, originating on healthy skin and resulting from a cutaneous infection by pus cocci. Only the *impetigos* and *ecthyma* will be discussed at this time, leaving the description of the follicular pyodermatitides to the chapter on the Folliculoses.

2. Several *chronic infectious dermatoses* are likewise pustular from the start, the pathogenic agent being capable of causing suppuration by itself alone (syphilis, tuberculosis, glanders, mycoses, etc.).

3. Some *eruptive fevers* are pustular at a certain stage of their course; this is true for *variola*, *vaccinia* and sometimes *varicella*. Cutaneous diphtheria may exceptionally assume the complete appearance of impetigo vulgaris. These diseases need only be mentioned in this connection.

4. Some *artifitial dermatitides* are, or may be, pustular from the start. Mercurial agents, fumes of tar and resinous plasters sometimes give rise to erythemas with small scattered miliary pustules; thapsia, tartrate of antimony, croton oil, etc., produce lenticular

pustules; oil of cade and analogous products sometimes give rise to papulo-pustules. Certain toxidermas of internal origin are likewise pustular (iodides, bromides). The question arises if these chemical substances are pyogenic in themselves, or if, as seems probable, their action is limited to favoring the penetration and activity of the pyococci. The point has not yet been settled. A special chapter (XXIII) will be devoted to these artificial dermatitides.

5. There still remain the *secondarily* and *accidentally pustular dermatoses*. When suppuration supervenes in the *eczematous* or *vesicular dermatoses*, this is evidently due to secondary infection by pyococci; secondary infection, however, has not been proved to be necessary in the *bullous* dermatoses, such as pemphigus and Duhring's disease, which includes a pustular variety.

The secondary suppurations will be referred to in connection with each of the dermatoses in which they are met, so that this chapter deals only with the *primary pyodermatitides* and a few *chronic infections*.

IMPETIGO.

The name *impetigo* belongs to an affection characterized by inoculable and auto-inoculable purulent bullæ, appearing rapidly on the healthy skin, drying in crusts which are often yellowish and meliceric, the underlying epidermis being simply eroded and healing in a short time without leaving scars.

Instead of developing on healthy skin, the suppuration and crusts which result from the drying of the pus may appear on a wound or on a pathological lesion, for instance eczema. These lesions are then said to be secondarily impetiginous, or better, *impetiginized*.

The idea of a microbic external pyococcic origin of impetigo and impetiginization dates back nearly to the first days of the discovery of bacteria. As regards the responsible bacterial species, investigators are divided into two schools, some regarding streptococci and others staphylococci as the pathogenic agents. An important advance was made when Sabouraud established the fact that the different clinical forms of impetigo are referable to different organisms.

There exist an *impetigo streptogenes*, an *impetigo staphylogenes*, and an *impetigo vulgaris*, in which the two microbic species are associated.

1. **Streptococcic Impetigo** or *Impetigo of Tilbury Fox*.—The primary eruptive lesion is a flaccid seropurulent blister, extending peripherally.

The blister from a hemp-seed to half a hazel-nut in size, appears in a few hours on a faintly pinkish base. Its fluid may be serous, stringy and at first hardly turbid, but it soon becomes cloudy and is transformed into sero-pus. The roof of the blister is a thin

opaline membrane, tense when the fluid is abundant, wrinkled and fluctuating in the opposite case.

In consequence of evaporation, or surface extension, the bulla becomes flaccid if it has not been so to begin with. It may rupture, allowing its fluid to escape; or it may dry in a crust in its center, while it enlarges peripherally, or more particularly on one side, through detachment of the horny epidermis. At this stage the bulla is always surrounded by a congestive areola. Extensive or serpiginous bullæ of this kind are met with measuring several centimeters in diameter.

Finally, the lesion shrivels up to a yellowish or brownish crust covering a red erosion; it lasts from four to eight days, then becomes detached, leaving behind it a pinkish or purplish macule, which persists a fairly long time.

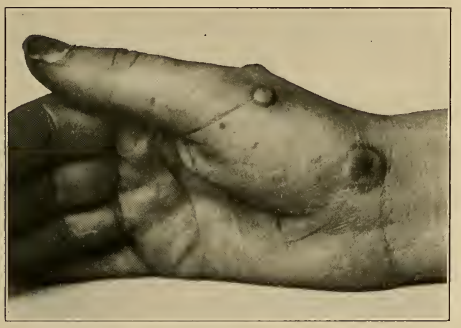


Fig. 45.—Coexistence of impetigo of Tilbury Fox on the wrist, and impetigo of Bockhardt at the root of the thumb, in a case of scabies.

The eruption is composed of a single bulla, or of more or less numerous bullæ, resulting from simultaneous or successive infections, or especially from spontaneous auto-inoculations. The bullæ are often in different stages of development.

Their seats of election are the face, especially around the mouth, the nose and ears, sometimes in the beard or on the scalp; furthermore, on the extremities, affecting the hands and fingers, or the feet, where they originate from excoriations caused by the shoes, etc.

The condition known as *whitlow* or subepidermic panaris, ["run-around"] is nothing more nor less than a bulla of streptococcic impetigo, derived from a fissure or a hang-nail, and having a tendency to extend around the nail.

Other tegumentary regions are more rarely affected, except in

case of gross neglect, traumatism, or scratching. In *scabies*, for example, complication with the impetigo of T. Fox is common, although less frequent than staphylococcic complications.

In the impetigo of T. Fox the glands belonging to the affected regions are usually swollen and tender, especially when the eruption is accompanied by *lymphangitis;* the absence of protective dressings, fatigue, overwork and general debility, favor this complication. These cases are associated with pain, fever, digestive disturbances, prostration; the local infection may also lead to a grave but fortunately rare general disease, a streptococcic septicemia.

In contradistinction to other forms of impetigo, the impetigo of T. Fox shows no predilection for a given age or sex.

FIG. 46.—Histology of impetigo of Tilbury Fox. Border of a very recent bulla of the ear. The cavity is the result of splitting of the epidermis at the level of the granular layer. The bulla which is subcorneal, contains a coagulated fibrino-albuminous fluid, detached epithelial cells, and a small number of pus cells. The rete which is not deformed and the edematous papillary body are seen to contain a few wandering cells which are beginning to make their appearance. × 90.

The affection lasts from three to eight days under suitable treatment and may persist for weeks or months under the opposite conditions, or when circumstances are present favoring autoinoculations.

The *pathological lesion* consists of a bullous elevation of the horny layers by a fluid composed of blood-plasm and leukocytes in variable proportion. The mucous body and the papillary body are infiltrated with more or less numerous wandering cells (Fig. 46).

The demonstration of the streptococcus, the primary pathogenic agent, is not easy in sections; it is made by cultures in ascites-broth in pipettes, according to Sabouraud's method, or by making numerous streak-cultures on agar slants with a single platinum wire

carrying a small amount of material, as recommended by Lewandowsky.

2. **Impetigo Vulgaris.**—This is the most frequent form of impetigo and is chiefly observed in children of all ages, especially from two to seven years, or in adults with a delicate skin. It is highly contagious and of polymicrobic origin.

It was formerly considered as the typical impetigo whose chief characteristic was said to be the thick, extensive, coarse, yellow crusts, which were described as meliceric on account of their resembling dried honey. The popular term of milk crusts is applied to this impetigo as well as to impetiginized infantile eczemas and seborrheal crusts, etc.

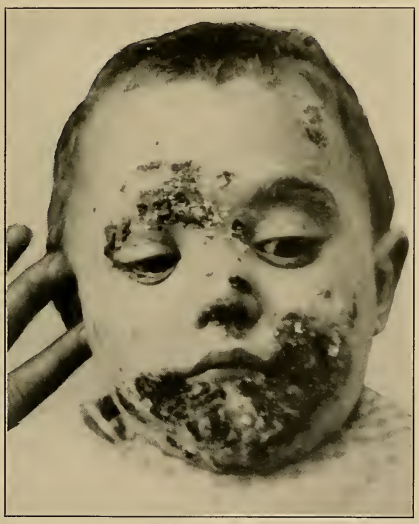


FIG. 47.—Impetigo vulgaris of the face in a child aged two years.

A crust can never be characteristic of a dermatosis as it is always merely a secondary lesion. On closely following a case of impetigo vulgaris, the initial lesion will usually be found to be a bulla of T. Fox impetigo; this rapidly becomes purulent, dries to a crust at its center while it extends at its periphery and in this way gives rise to *circinate crusted lesions*. At the circumference appear either similar lesions, or staphylococcic pustules, such as will be described further on. The glands are usually enlarged.

The eruption often takes its origin near the nostrils, following coryza; near the mouth, following fissures of the lips or "perlèche" (Fig. 47), near the eyelids in the case of conjunctivitis; on the ears, in case of suppurative otitis; in the occipital region of the scalp in children and women having head lice; on the beard, where it is inoculated by means of the razor (Fig. 48). [In my experience impetigo in children is almost invariably associated with pediculosis capitis.]

FIG. 48.—Impetigo vulgaris, derived from inoculation with a razor.

Impetigo larvalis is the term applied to an eruption which covers the face like a mask; *impetigo granulata* affects the scalp or the beard, giving rise to crusts whose fragments adhere to the hairs. Other varieties have been described as *impetigo sparsa*, *figurata*, etc.

It is not nearly so important to fit the exact descriptive term to the objective appearance as to determine if the impetigo is primary or secondary in character. As a matter of fact, it may point the way to an infection of the mucous membranes or body orifices as stated above, or complicate an eczema, a burn, or some traumatic dermatitis, scabies, lupus (Fig. 169), syphilides, etc. These impetiginized

eruptions are sometimes not clearly recognized until after several days of treatment.

The microbes of impetigo vulgaris may become implanted and vegetate wherever they find a point of entrance.

The term *impetigo contagiosa* has no special significance and applies to cases where genuine epidemics are observed in families or schools.

Impetigo has a marked tendency to recurrences which are explained by the persistence of virulent pyococci in the lesions, called by Sabouraud the *chronic remains* of impetigo, such as redness and crust-formation behind the ears, around the nostrils, perlèche, blepharitis and styes, and even pityriasis simplex (dry seborrhea). These chronic infections and the adenopathies dependent upon them enter into the clinical picture of scrofula.

Impetigo of the Mucous Membranes.—When astride the free border of the lips, impetigo is crusted in its cutaneous portion and diphtheroid in its mucous portion. Sevestre and Gaston have described an *impetiginous stomatitis*, the characteristics of which are as follows: Diphtheroid spots of a yellowish white color; imbedded and incorporated in the epithelium, scattered over the mucosa of the lips, the cheeks, sometimes on the tongue and palate, never on the isthmus of the pharynx or in the throat; usually, ulceration of the alveolar margin; a low degree of contagiousness; ordinarily associated with impetigo of the face. Failure to recognize this affection may result in the most annoying errors in diagnosis.

Certain coryzas, pyococcal blepharo-conjunctivitis, even phlyctenular conjunctivitis, may, strictly speaking, be interpreted as impetigo of mucous membranes.

3. **Staphylococcic Impetigo,** or *Impetigo of Bockhardt.*—This is characterized by a primary pustule, containing yellowish creamy pus, often centered by a hair and surrounded by a congestive areola. The lesion has the size of a pin-head to that of a large lentil; the pus collects under the horny layer, elevating and distending it (Fig. 49).

A perifollicular seat is common but not constant and Bockhardt's impetigo has so many points of contact with impetigo vulgaris that it must be grouped with the latter rather than with the folliculitis. The pustules are usually multiple, often very numerous and are grouped in one or more regions whence they may spread. The eruption has no preference for special parts of the body, but develops at any point where the horny layers have ceased to oppose an efficient barrier against the penetration of the pyococci. This occurs in cases of traumatism, scabies, chemical dermatitis, or epidermal maceration through the application of a simple poultice or plaster. Preëxisting suppurations, dirt, pruritus and neglect, constitute

conditions favorable to its development. A youthful age, lymphatism, overexertion, etc., seem to act as predisposing factors.

The pustules of staphylococcic impetigo appear in a few hours, being more resistant than those of the preceding forms; they rupture late or accidentally. The pus is then thickened into yellow crusts. Unopened pustules shrivel up first in the center and are cast off by eviction.

Bockhardt's impetigo is accordingly merely one of the elementary forms of *cutaneous staphylococcia*, the most superficial and most benign type of the disease. There exist intermediary forms between the impetigo of Bockhardt and the more or less deep folliculitides (Plate II). Combinations with other forms of staphylococcia frequently occur.

FIG. 49.—Histology of the pustule of Bockhardt's impetigo. The cavity of the pustule is situated within the rete; its roof is formed by the horny layer, doubled in places by the granular layer; its floor is represented by the lower strata of the rete, which are seen to be crowded and flattened. The oblique section of two downy hairs may be seen floating in the purulent contents of the pustule. × 60.

A bullous form of impetigo, generally known under the name of *epidemic pemphigus of the newborn* will be discussed further on p. 178.

The affection described under the name of *infantile vacciniform dermatitis* by Hallopeau, *vacciniform herpes*, by Fournier, is probably merely a variety of impetigo. It appears in the form of erythematous spots, the epidermis of which is rapidly raised by a turbid fluid, with central umbilication. It is observed in neglected infants, especially around the ano-genital region. The eruption may simulate varicella.

Treatment.—Every practitioner should know how to treat impetigo. Whether it be primary or secondary, the treatment is the same, but in the latter case, after the complication has been removed, the original dermatosis will require attention.

In the first place, the treatment must be local. A fundamental rule is to get rid at once of all crusts or active inflammation that

may be present. This will be accomplished in a day or two by means of moist softening or slightly antiseptic dressings, sprays or starchy poultices, to be applied cold. At each dressing, at least three times in twenty-four hours, antiseptic or astringent lotions must be applied; the solution which for some time has achieved, or rather resumed general and well deserved favor is Alibour water, the formula of which dates back to the reign of Henri IV. As soon as the surfaces are cleansed, pastes or salves made with boric acid, camphor, white precipitate and especially yellow precipitate are indicated. A mercurial plaster is serviceable for the isolation of scattered lesions of impetigo. Camphorated lotions or ichthyol pastes help to prevent recurrences.

Internal treatment, contrary to a commonly held view, is by no means indispensable, and *must never be considered as sufficient by itself alone.* It may be necessary sometimes to prescribe cod-liver oil, iron iodide, arsenic, or the ferments and yeasts. Dietetic measures, residence by the seaside, or mineral springs, influence only the general condition.

ECTHYMA.

The meaning of the term ecthyma has undergone some slight changes, according to different authors and at different times, so that it is somewhat difficult to separate ecthyma from the impetigos, from rupia of the older writers and from ulcers of the skin.

Ecthyma actually represents a *pustulo-ulcerative pyodermatitis;* namely, a microbic dermatitis of external origin (XXVI) like impetigo, beginning like the latter with a pustule, but differing from it by the greater dimensions of its lesions and especially by their ulcerative character. The ulceration is frequently covered by a crust, which may be rupioid. Ecthyma always leaves a scar behind it. Like all other pyodermatitides, it is inoculable and auto-inoculable. Its special clinical characteristics are referable to a peculiar virulence of its pathogenic agents, or to a deeper inoculation of these agents, favored by some tissue lesion or preliminary affection, or finally to the soil on which it develops.

When the mode of formation of an ecthyma lesion can be watched, it is seen to begin with a *pustule,* usually flattened and with turbed contents, like the impetigo of T. Fox, more rarely tense and containing a creamy pus, like the impetigo of Bockhardt.

This pustule, having very rapidly reached nummular dimensions, shrivels up into a more or less thickened, yellowish or brown, adherent *crust,* which is flattened or protuberant, sometimes ostreaceous, surrounded at first by a bullous collar and always by a congestive halo. When this crust falls off or is removed by means

of the dressings, it is seen to cover a rounded or oval ulceration, more or less deeply encroaching upon the derma. Its borders are regular and clean-cut; the floor is red or pultaceous, sloping toward the center in the stage of advance, granulating in the stage of repair; the secretion consists of viscid or clotted brownish and blood-tinged pus; the base is not indurated, but sometimes diffusely edematous.

After lasting two or three weeks in favorable cases, but much longer under bad conditions, the ulceration heals through granulation and *cicatrization;* the scars are often pigmented at their border.

Glandular enlargement, lymphangitis, phlebitis, abscesses, are rare complications.

The lesions are usually multiple, rarely very numerous and, as a rule, of different ages, having originated from successive autoinoculations.

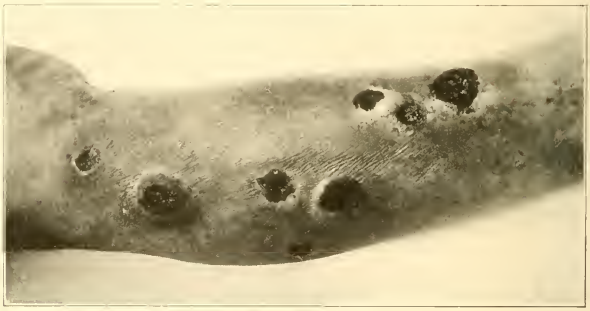


FIG. 50.—Ecthyma of the leg. The lesions have been deprived of their crusts and cleansed by means of moist dressings.

Ecthyma has its *seat of election* on the lower limbs (Fig. 50); the buttocks and the back are less frequently affected, the part played by congestion and circulatory stasis is therefore very evident.

Ecthyma is usually observed only during the first half of life; it affects overstrained, weakened, scrofulous, diabetic, varicose, or alcoholic subjects; not uncommonly, it is induced by scratching due to uncleanliness, or by scabies and lice. Ecthyma has figured to a great extent among the soldiers in the trenches.

There are no valid reasons for distinguishing a *cachectic ecthyma*, a *scrofulous ecthyma*, etc., nor is it correct to describe as *scabies-ecthyma* all the pyodermatitides complicating the itch. The antiquated terms of *syphilitic ecthyma* and *syphilitic rupia*, to designate ulcerative syphilides, are altogether objectionable.

In the presence of unfavorable nutritional conditions and local circulatory disturbances, an ecthyma pustule may become the starting-point of a leg-ulcer.

The **pathological anatomy** of ecthyma, in the ulcerative stage, shows a deep and remarkably distinct ulcer, bordered by a thin layer of leukocyte infiltration; apparently resulting from molecular disintegration and not from sloughing. The pus contains tissue-debris, elastic fibers, altered red corpuscles, and various cocci.

Ecthyma is ascribed by authors either to the streptococcus or to a microbic association. Vidal's statement that experimental inoculation of ecthyma-pus will produce this dermatosis, but no other pyodermatitides, is not strictly correct; however, the ecthyma-eruption

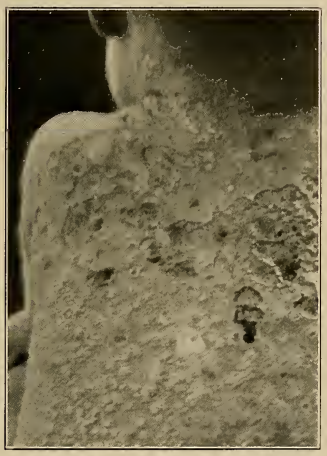


Fig. 51.—Erythema terebrans of the back in a child aged two and a half years.

is not infrequently pure and is not associated with other pyococcic manifestations. According to recent investigations of Lewandowsky ecthyma seems to be a simple streptococcia, closely related to the impetigo of T. Fox and with a slight tendency to become secondarily infected.

The name *ecthyma terebrans* is applied to a pustulo-ulcerative eruption in children, rare and rather obstinate, occupying the buttocks, thighs, and back progressing by contiguity, in the form of lenticular, sometimes vacciniform or even gangrenous pustules (*gangrenous varicella* of English and American writers). These pustules become confluent and give rise to enormous polycyclic

ulcerations, with festooned margins, a grayish floor and dusky circumference, leaving honey-combed cicatrices (Fig. 51). This grave eruption has been observed especially in weakly newborn infants suffering from diarrhea (Neumann's *ecthyma of cachectics*); exceptionally also in older children and even in adults. According to Bosellini and others, the affection is due to the bacillus pyocyaneus.

The **treatment** of any ecthyma of the lower limbs requires rest in bed, moist applications to loosen the crusts, followed by lotions of Alibour water or peroxide water; the ulcers are then dressed with a salve or paste of yellow oxide, or an absorbent or antiseptic powder may be used, such as dermatol, aristol, etc. Touching with a silver nitrate solution may prove useful. During the stage of repair, the ulcerations are advantageously covered with red oxide ointment or balsam of Peru.

General treatment is not usually necessary; regulation of the patient's hygiene will suffice.

PUSTULES OF THE INFECTIOUS CHRONIC DERMATOSES.

Syphilis, tuberculosis, glanders and certain mycoses and tropical diseases may give rise to *pustular syndromes*.

Syphilis. — There exist syphilides which are *only apparently* pustular; no drop of pus can be obtained by puncturing them with a needle. Such syphilides are known as *papulo-crusted* or *impetiginous syphilides*. They are generally mixed in with a crop of lenticular papules, of which they are merely a variety; or they may predominate in a given eruption.

The more or less profuse eruption is irregularly scattered over the trunk, limbs, face and scalp; it consists of round lenticular crusts, brownish-yellow, swollen, slightly adherent, sometimes exuberant, ostreaceous or rupioid (see Fig. 94), which cover a papule with a smooth moist surface, instead of an ulceration, as one might be led to believe.

Precocious malignant syphilides of the ecthymatous type begin, before undergoing ulceration, with a large papule the epidermis of which is raised by pus; this hardens into a crust; under this crust and at its periphery the ulceration develops and progresses.

Tuberculo-crusted syphilides, which are apt to be circinate, are only transitorily pustular; they belong rather to the tuberculo-ulcerative eruptions.

Tuberculosis. — Tuberculous ulcers begin as very small papulo-pustules which open and coalesce. In *tuberculosis verrucosa*, deep pustules are almost regularly seen, the contents of which can be squeezed out by compressing the vegetating patch between the fingers.

There exists a form of lupus exedens which is entitled to the name of *pustular lupus*.

It is especially important to keep in mind the very frequent occurrence of impetiginous changes in eroded lesions of lupus; fungoid tubercles, open scrofulous gummas, or atypical tuberculous ulcers becoming covered with yellow or brownish crusts. A diagnosis of impetigo or ecthyma must be carefully excluded in such cases. In the absence of information regarding the development, it suffices to detach or otherwise remove the crusts, to bring into view the grave underlying lesions.

Papulo-necrotic tuberculides are genuine pustules at a certain stage of their development. They are generally recognized by their very peculiar course and sometimes by their topographical distribution.

Glanders.—One of the clinical features of acute glanders consists in a pustular eruption resembling that of smallpox. The round, non-umbilicated pustules form rapidly, occupying especially the face, the mucous membranes and the limbs; they rupture later on, leaving extensive ulcerations. The general symptoms suggest the diagnosis which must be confirmed by bacteriological methods.

The *mycoses* (sporotrichosis, etc.), *Leishmaniasis* (Biskra boil or button) and *verruga Peruviana* may give rise to pustules at their onset, or in the course of the disease (XXIX).

CHAPTER X.

BULLÆ AND BULLOUS DERMATOSES.

Bullæ or phlyctenes (blebs) are circumscribed elevations of the epidermis containing an ordinarily clear and serous, sometimes turbid or hemorrhagic fluid; when the fluid consists of pus, the lesion becomes a *purulent bulla*.

Bullæ differ from vesicles by their generally larger size as well as by their structure and mode of formation. Their shape is round or oval and their size varies from that of a pin-head to that of a hen's egg or larger; their surface is tense or flaccid. They terminate through rupture, suppuration, or simple desiccation; in all these cases, they are followed by a crust of variable color and thickness according to the character of the exudate, covering a more or less deep erosion. The crust becomes detached in five to fifteen days, leaving almost invariably a temporary red or brownish macule in its place.

Blebs or bullæ do not result from a progressive process, like vesicles, but from a genuine splitting of the epidermis. Their cavity is therefore unilocular from the start; when the fluid contents have been emptied, through pricking or tearing the bulla, it collapses entirely.

Two processes are concerned separately or jointly in the formation of bullæ. Most frequently they follow a local dermic edema, the edematous fluid under high pressure filtering through the Malpighian layer, until it is arrested by the horny layer which it raises by accumulating below it. This results in the production of a *superficial* or *subcorneal bulla* (Figs. 46 and 58).

In other cases, the still more sudden rush of fluid detaches the epithelium as a whole, giving rise to a *deep* or *subepithelial bulla* (Fig. 56.)

In case of the second mode of bleb-formation, the mutual cohesion of the cells of the mucous body is pathologically diminished; the intercellular filaments have lost their resistance; they separate under the influence of the slightest excess of pressure of the intercellular plasma. This abnormal condition is designated as *acantholysis*, after Auspitz, and the resulting bullæ are known as *acantholytic bullæ*.

Bullous Dermatoses.—The group of bullous dermatoses is very extensive and very complex. The older writers interpreted all

bullous eruptions as *pemphigus,* a circumstance which resulted in extraordinary and persistent confusion. This designation is now reserved for a small minority of these cases. The following eruptions do not belong under the heading of pemphigus:

Traumatic bullæ, or blisters, produced by strong pressure [or friction]; and those which result from burns, or from the action of caustics and vesicants. These are artificial external dermatitides of bullous form and will be discussed further on (XXIII).

Bullæ, occurring as *epiphenomena* in the course of definite nervous or infectious diseases, such as syringomyelia, leprosy, purulent infection, etc.

Accidentally bullous dermatoses, such as erysipelas, eczema, dysidrosis, ichthyosiform hyperkeratosis; mention must be made of bullous urticaria, bullous polymorphous erythema or hydroa, bullous syphilides and bullous toxicodermas of internal origin.

External microbic bullous eruptions, which according to Unna are all entitled to the name of *impetigo.* Although it remains doubtful if certain pathological varieties should be grouped under this heading rather than with acute pemphigus, this seems to be justifiable as regards the so-called *epidemic pemphigus of the newborn* and of *adults,* which is really a bullous impetigo.

Eliminating these forms there are left some essentially bullous affections, of undetermined but decidedly variable type, which are designated as *pemphigus,* and include: (1) Pemphigus acutus febrilis gravis. (2) Recurrent polymorphous pemphigus, or Duhring's dermatitis; (3) Pemphigus chronicus; (4) Pemphigus foliaceous; (5) Pemphigus vegetans; (6) Pemphigus congenitalis. So-called pemphigus hystericus will later be considered in a few lines.

Accidentally Bullous Dermatoses.—Some of these require special mention on account of the difficulties of diagnosis and interpretation which they may occasion.

Bullous Urticaria.—This is a rare variety of urticaria in which all or some of the lesions become crowned with a bullous prominence, followed by a crust. The eruption may be chronic or recurrent. In view of the pruritus and the erythematous base of the bullæ, this affection may be confused with Duhring's dermatitis, from which it is distinguished by its irregular distribution and the absence of true polymorphism, each bulla originating on an urticarial wheal. Sometimes, it is possible to demonstrate a tendency to urticaria in the patients.

Bullous Polymorphous Erythema or Hydroa.—In certain cases of polymorphous erythema, localized as usual on the back of the hands, on the wrists, elbows, knees, on the face and especially on the forehead, etc. (Fig. 52), some lesions or most of them may become the seat of vesicles or of tense bullæ which when pricked with a

pin void a lemon-yellow or reddish-fluid. The bullæ occupy the entire surface of the papular elevations or only their center; sometimes they are situated at the periphery.

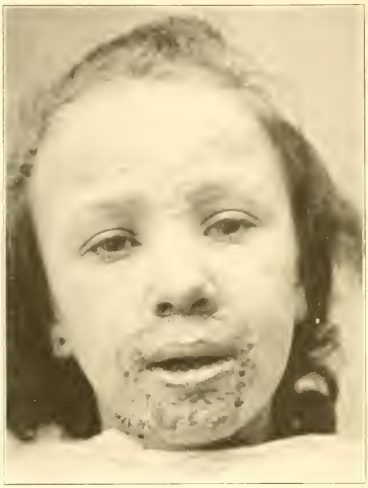


Fig. 52.—Erythema bullosum; first attack, in a girl aged eleven years. (The buccal mucosa were affected at several points.)

Fig. 53.—Erythema bullosum of the variety hydroa vesiculare of Bazin, or herpes iris of Bateman.

The name of *Bazin's* **hydroa**, or **herpes iris** of Bateman, is applied to a variety in which the lesions, consisting of a small bulla or central crust, surrounded by a bright red or purplish disk, a vesiculobullous circlet and narrow erythematous border, present a striking cockade-like arrangement (Fig. 53).

The eruption often affects also the lips, the mouth, the tongue, the pharynx and the other mucous membranes.

Hydroa buccale, characterized by bullous elevations which are rapidly replaced by very painful nummular deep red or diphtheroid erosions, simulates and is sometimes mistaken for mucous patches. In exceptional cases it may be accompanied by fever and infectious symptoms, with severe visceral complications.

Polymorphous bullous erythema usually lasts from two to five weeks; it may be prolonged beyond that time by successive eruptions, or it may recur at variable intervals in a number of attacks. This tendency to relapse and the unusual severity of the sensations of itching and heat, in some cases result in so close a resemblance to Duhring's disease as to render the diagnosis necessarily doubtful.

Bullous Syphilides.—The eruption known under the name of *syphilitic pemphigus* is met with on the palmar and plantar regions of newborn children with congenital syphilis. It consists of papular lesions of a purplish or coppery hue, discrete or confluent, the epidermis of which is raised by a turbid or blood-stained fluid; the dimensions of the bullæ vary from those of a hempseed to those of a large bean, but confluence in extensive patches may occur.

In two or three days, these bullæ dry into crusts, covering an ulceration. Their fluid contents and especially the fluid obtained by scraping their floor, contain enormous numbers of spirochetes. It is exceptional for the eruption to become generalized under the same form; but a few aberrant bullæ, or syphilides of another type, may be found in other portions of the body.

This eruption, the only syphilide assuming a bullous form, appears almost exclusively at the time of birth, a few days before or after. It is pathognomonic.

Bullous Toxicodermas.—*Antipyrin* bullæ represent a transformation of the erythematous patches produced by antipyrin in some individuals (Fig. 145). They originate abruptly in any region, especially the genitals and the mouth, reappearing with each repeated administration of the drug and leave a brown discoloration.

Iododerma bullosum is a rare affection produced by potassium iodide or its congeners; the lesions may be clear, distinctly pemphigoid bullæ, of rapid development, located especially on the neck or in the folds (Fig. 146); or the condition may consist of very rapidly purulent and extensive bullæ, the center of which becomes fungoid and crusted, situated in very variable numbers on the face, the mouth, the limbs or the trunk; they suggest the fungoid syphilides or more particularly pemphigus vegetans. This eruption may persist during several weeks, especially if the medication is continued.

Bromides, arsenic, etc., are likewise capable of causing the appearance of blisters.

BULLOUS IMPETIGOS.

The impetigos are pyodermatitides the eruptive lesion of which is a pustule or a purulent bulla; they are all more or less contagious.

A somewhat distinct bullous type is generally described under the name of *epidemic pemphigus of the newborn*. It is encountered in nursing infants, in asylums, hospitals, or even in families—more rarely in adults—in the form of an acute eruption of clear tense hemispherical bullæ, the size of a lentil to that of a nut; from one to about thirty in number. They occur in the folds of the neck, trunk and limbs, rarely on the face, but are never encountered in the palmar and plantar regions.

These bullæ supervene in healthy or in marasmic children, in successive crops, in the form of red spots which very rapidly become blisters; at the end of a few hours the bulla ruptures and the thin crust falls off in a few days. Recovery is the rule; exceptionally, in weakened patients, symptoms of severe general infection have been noted.

FIG. 54.—Impetigo bullosum in a child aged sixteen months.

This affection is highly contagious. Vidal has shown that it is possible to inoculate and auto-inoculate the fluid of the bullæ. It contains, according to Peter, the Staphylococcus aureus, some albus and a special diplococcus which is by several authors considered the cause of the disease; perhaps, in addition, a streptococcus. The Staphylococcus aureus is regarded as the real pathogenic agent by Dohi and Jadassohn.

Bullous impetigo is sometimes observed also in older children (infantile pemphigoid of Jadassohn) in combination with various pyodermatitides (Fig. 54).

Confusion must be avoided with varicella, generalized vaccinia, impetigo vulgaris and the bullous syphilides. However, the features

of these various eruptions are usually sufficiently distinct to guard against errors in diagnosis.

The *treatment* consists in extreme cleanliness, absolute hygiene and isolation of the patient. Locally, washing with Alibour water will suffice, and dressings with a bland powder, an oil and lime liniment, or yellow precipitate salve.

PEMPHIGUS ACUTUS FEBRILIS GRAVIS.

This is a systemic infectious disease, with a bullous eruption, studied by Nodet (1880), George Pernet (1895–1896) and Brocq; it affects almost exclusively butchers, sausage-makers, pork-choppers, tanners, cooks, etc., namely persons who handle dead animals. The infection usually follows upon some injury of the hand, manifesting itself, abruptly by chills, prostration, vomiting or diarrhea, delirium or depression and a temperature of 40° (104°). The eruption appears twenty-four to forty-eight hours later, in the form of tense bullæ with yellowish or hemorrhagic contents; originating on red spots and almost invariably undergoing rupture. It occupies the neck, the chest, the limbs or the entire body; discrete at first, it soon becomes confluent and may involve the mucous membranes. Death results in over three-fourths of the cases, at the end of one to three weeks, under typhoid symptoms, albuminuria, various congestions, etc. Recovery takes place through gradual subsidence of the symptoms in three to six weeks.

The anatomical lesions of acute pemphigus and its pathogenic microbe are unknown. The condition evidently represents a variety of septicemia.

The indications are to combat the infection by means of quinine, collargol, serum injections and intestinal irrigation, and to support the organism by all possible means. Moist or oily dressings, cotton wraps, and so forth serve to relieve the burning sensation which is often extremely distressing.

RECURRENT PEMPHIGUS, DUHRING'S DISEASE OR POLYMORPHOUS DERMATITIS (BROCQ).

A well-characterized dermatosis in typical cases was described by Duhring, in 1884, under the name of *dermatitis herpetiformis.*

This affection, which the Vienna School confused with chronic pemphigus and multiform erythema, was designated by the old French writers as *pemphigus with small bullæ,* or *pruriginous pemphigus,* or "*arthritide bulleuse*" (Bazin). English authors, following Tilbury Fox and Colcott Fox, prefer the name of *hydroa herpetiforme.*

In the opinion of Brocq, who thoroughly investigated this question, the disease is entirely distinct from pemphigus; and since the eruption is far from being invariably herpetiform, he extended the scope proposed by Duhring and established a group of painful polymorphous dermatitides [dermatites polymorphes douloureuses] comprising numerous forms or varieties. This view is becoming more and more generally adopted.

Personally, like many others, I put together under the name of Duhring's disease all the varieties of the disease, of which the American author originally described only the form with small vesicular lesions. Instead of attempting to multiply the clinical types, according to the dimensions of the bullæ, etc., I shall endeavor to map out a general view, in the following description:

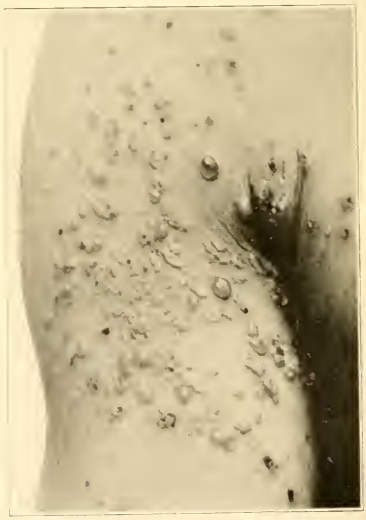


FIG. 55.—Dermatitis of Duhring. Anterior aspect of arm and right axilla.

Symptoms.— Four clinical features are characteristic of Duhring's disease: (1) The polymorphism of the eruption; (2) painful phenomena, usually very pronounced; (3) the usual preservation of good general health; (4) the tendency to recurrences. [The tetrad of symptoms which seem most characteristic are: The polymorphism of the lesions; their more or less marked herpetic grouping; the pruritus and the tendency to recurrences. Sensations of pain

are noted in the minority of the cases in my experience.] The *onset* is marked sometimes by the eruption, sometimes by a preliminary pruritus.

1. The *eruption* is polymorphous and presents itself under very variegated aspects (Fig. 55); it often covers a large portion of the limbs and the body. It is composed of erythematous patches, papules, vesicles, bullæ and sometimes pustules.

The multiple spots or erythematous patches are often urticarial and marginated; in the same case, they may have nummular dimensions or resemble large polycyclic surfaces; more rarely, simple papules are met with.

A tendency to bleb-formation manifests itself early through the appearance, on the surface of the patches, of sometimes uniform, herpetiform, grouped or scattered vesicles, often arranged as a border; or bullæ may be seen, from the size of a pea to that of a nut, with clear or rapidly purulent contents. Vesicles or bullæ may also originate in the healthy skin in the vicinity of the patches, or they may swarm out to a distance. In a general way, the figured erythematous patches, interspersed with herpetiform vesicles or bullæ are the most characteristic feature.

The most frequent initial localization is on the limbs, especially on the forearms; but the eruption may start anywhere. It proceeds by enlargement of the primary lesions and the production of new lesions, appearing every two or three days in small numbers, or in large crops every five to ten days. The duration of each lesion is limited, however. The erythema fades; the vesicles and bullæ burst through scratching and are replaced by red surfaces, raw or covered with crusts; the latter fall off and leave pigmentary macules, very rarely cicatrices. Lesions of different age are thus present at the same time.

As a rule, the eruption is decidedly symmetrical. It affects especially the limbs, the buttocks, the chest, etc., but has a tendency to cover the entire body; possibly the face, the scalp, the palms of the hands and the soles of the feet are less frequently involved. When the last-named regions become keratotic, arsenic medication may possibly be responsible.

The mucous membranes, more particularly the buccal mucosa, are affected in nearly one-half of the cases; the lesions here resemble those of hydroa. [Involvement of the mucosa is by no means so frequent in this country as in the author's experience.]

An endless number of eruptive varieties might be described; but it is enough to point out that the predominant lesion may take the form of erythema, or of herpetiform vesicles, these being the cases which Duhring had in mind, or of bullæ, which led Kaposi and his school to see in this affection merely a modified form of pemphigus

vulgaris. American and English writers have described a pustular variety, with pustules from the onset. Sometimes the bullæ are extensive and plainly vegetative in character.

Cases are observed in which the eruption is localized in a certain region of the body; other cases in which polymorphism is absent, notably in children or youthful individuals, vesicles or bullæ occurring exclusively. In still other cases, on the contrary, nothing is seen but patches of marginate erythema, incessantly recurrent and pruritic, with very scanty vesiculation.

2. The painful phenomena are one of the fundamental characteristics of Duhring's disease, but cannot be considered as constant and pathognomonic, as claimed by Besnier and Brocq. They consist of sensations of itching, heat, burning or acute pain. They may precede the eruption by a few days, but are especially liable to accompany each crop, with exacerbations in the evening and during the night. Although usually of moderate severity, the patient's sufferings may reach an intolerable degree, and have even led to suicide.

3. The general condition, notwithstanding the very extensive cutaneous lesions, the pruritus and the insomnia remains remarkably good, the patients eating and digesting well and losing no flesh. In exceptional cases attacks of diarrhea or a slight rise of temperature have been noted. The visceral complications (pulmonary and especially renal) which have been said to occur, are probably referable to intercurrent diseases.

4. The *course* is variable. Attacks as described above last from six weeks to three months, sometimes six months or a year. But they are often followed by a subsidence, or even a return to normal, lasting several weeks, several months or even a year or longer, then a new attack supervenes, and so on at irregular intervals, up to ten or fifteen times. Finally, the attacks become milder and ultimately cease. The proportion of recoveries can hardly be specified.

The disease is sometimes prolonged until death, which in rare cases is due to pemphigus foliaceus or to cachexia: much more frequently to an intercurrent affection. Hence, although Duhring's disease must be considered as very grave, this is rather on account of its long duration, the suffering caused by it and the interference with social relations which it entails than on account of a risk to life.

Its practically constant tendency to proceed not only in small distinct or continuous crops, but in attacks or recurrences separated by periods of subsidence, seems to me to constitute one of its most characteristic features.

Cases with identical symptoms are observed, however, in which the disease is restricted to a single attack, mild or severe. These

may be classified under polymorphous erythema. Brocq admits a group of acute non-recurrent painful polymorphous dermatitis.

Pathological Anatomy.—The erythematous plaques are composed of congestion, with well-marked edema and abundant diapedesis in the papillary body; eosinophile cells are present in large numbers.

The bullæ form as the result of subepidermic rupture (Fig. 56), sometimes subcorneal rupture. Occasionally they result from enlargement of interstitial vesicles. Deep or superficial bleb-formation is accordingly observed in this disease together with vesiculation.

The fluid of the vesicles or bullæ contains at first a large preponderance of eosinophiles, in a proportion of 30 to 95 per cent. The blood usually shows from 12 to 15 eosinophiles in 100 white corpuscles, sometimes only 5 per cent., exceptionally up to 30 per cent. There are no constant visceral or nervous lesions.

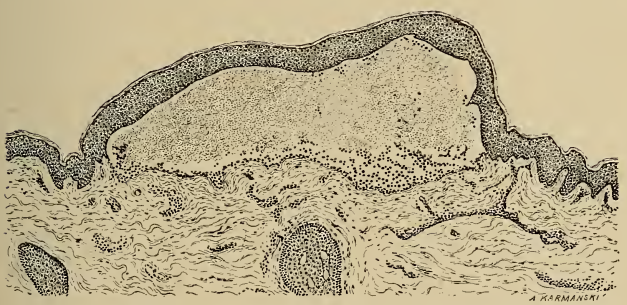


Fig. 56.—Histology of Duhring's disease. Section of one of the bullæ represented in Fig. 55. Note that the bulla is subepidermic; the epidermis has been raised as a whole by the fluid exudate. Among the white corpuscles seen at the floor of the cavity or scattered in the cutis are many eosinophile cells. × 50.

Etiology and Character.—Although a rare affection, Duhring's disease is nevertheless the most common bullous disease of the pemphigus group. It seems to be more widely distributed in England and in America. It is observed in very young children, in youthful, middle-aged or old individuals, in both sexes alike. [In American dermatological practice it is seen in the ratio of one case out of five hundred.]

The etiology is unknown and the cause has been sought in a neuropathic constitution, overwork, emotional disturbances, alimentary or medicinal intoxications. The disease is not at all contagious nor can it be inoculated. Two principal theories are held by the majority of writers: Some believe that endogenous or exogenous

toxins of microbic or other origin act upon the nervous system, causing the pruritus and the eruption. In the opinion of others the eosinophilia, which in this disease exists in the blood as well as in the cutaneous lesions (as shown by the investigations of Leredde and Ch. Perrin), indicates its hematogenous origin (hematoderma), the bone-marrow being probably primarily affected.

Eosinophilia, the significance of which is, moreover, imperfectly understood, may, however, be absent in Duhring's disease, whereas, on the contrary, it sometimes [commonly] exists in other bullous affections, pemphigus foliaceus, pemphigus vegetans and even exceptionally in chronic pemphigus, as I have been able to demonstrate.

Treatment.—All lotions and applications in common use for itching affections have been tried, with variable results. Some cases may derive benefit from the employment of absorbent powders, or moist dressings, which, however, tend to macerate the epidermis; or from the application of lime-and-oil liniment or various creams; while others do well under simple pastes, or even sulphur or tar pastes. Large bullæ should be pierced with a flamed needle; the excoriations are dressed with naphtalan, cold cream or lanolin. In case of very extensive lesions where the dressing exhausts the patient too much, he may be placed between two sheets in a thick layer of talcum powder (dry treatment).

General Treatment.—The most popular internal treatment is arsenic, in progressive long-continued doses, administered in hypodermic injections, if necessary. Novarsenobenzol has been found to be very efficacious in certain cases. Balzer advocates adrenalin. I have sometimes obtained remarkable but very unreliable results from repeated injections of physiological salt solution or isotonic sea-water, or sugar solutions, hypertonic or not. It must be kept in mind that hypodermic injections in large doses, and still more so intravenous injections, with or without preliminary bloodletting, may give rise to a well-marked febrile reaction. It is desirable to further investigate this mode of medication, as well as serotherapy with organic sera in its different forms (autoserotherapy, etc.). [On the subject of autoserotherapy the views of American authors are greatly divergent; a few speak enthusiastically of its value, others find it of no use.]

In all cases a very strict diet must be prescribed, free from stimulants, preferably a milk diet, and all organic functions must be carefully supervised.

Rare Dermatoses Related to Duhring's Disease.—Under this heading I have grouped a number of pathological forms which have received special names, but are perhaps merely varieties of the disease described above. This remark is certainly true of **herpes**

gestationis of Milton and Duncan Bulkley. This condition is really a Duhring's disease, with the special characteristic of developing in the course of pregnancy, in the fifth to sixth month, or sometimes after delivery. The attack lasts a few weeks to several months. Recurrence is the rule with each succeeding pregnancy, each attack occurring earlier in the course of the gestation and lasting longer.

Hydroa puerporum of Unna is in all probability likewise a form of Duhring's disease, appearing in young children in the form of acute polymorphous attacks, recurring especially in the summer and disappearing around the age of puberty.

Hydroa Vacciniforme of Bazin, or *Summer eruption* of Hutchinson, seems to be an altogether different kind of affection. The eruption, sometimes preceded by general malaise, accompanied by a burning sensation and local tension, is not polymorphous, but vesiculo-bullous. At first tense and prominent, the small lenticular bullæ spread out, become umbilicated, attain the size of a finger-nail, become transformed into brownish pustules with a central depression and dry in crusts which leave cicatrices resembling the pits of smallpox. Central necrosis is occasionally noted.

The eruption occupies the exposed regions, especially the cheeks, the nose, the ears and the hands, being very rarely found elsewhere. It appears in the spring, under the influence of exposure to light, notably short-wave rays, as has been demonstrated experimentally. The patients, as a rule, have hematoporphyrinuria. The affection begins in the first few years of life, and usually ceases spontaneously at the age of twenty to thirty years.

There is accordingly a hypersusceptibility to light radiations, due to abnormal tissue-juices; it is an established fact that hematoporphyrin sensitizes toward the action of light.

As to the relations of **Impetigo herpetiformis** of Hebra-Kaposi, studied in France by Dubreuilh, with herpes gestationis and Duhring's disease great uncertainty prevails. This very rare affection is practically restricted to pregnant women, although a few cases are said to have been observed in men.

The eruption consists of red and swollen nummular spots which became covered with small miliary pustules, increase in size and became confluent in large surfaces, crusted in the center and pustular at the periphery. It usually starts in the inguino-crural region, the umbilicus, the loins or axillæ, and may become generalized, even involving the mucosæ. Severe general symptoms are present, in the form of chills, remittent fever, a typhoid condition, tetany, eclampsia. In 19 of 84 cases collected by Borzecki, the outcome was death.

No cultures can be grown on ordinary media from the contents of the pustules. If the condition is not a septicemia, but another

pregnancy auto-intoxication, as in herpes gestationis, it would be reasonable to try injections of serum from normal pregnant women.

It is also not known at the present writing if the *continuous acrodermatitis* of Hallopeau and others, or *recurrent phlyctenoses of the extremities* of Audry, are pyodermatitides favored by a predisposed trophoneurotic territory, or clinical forms of Duhring's disease. The affection begins, at any age, under the guise of a whitlow; but new purulent bullæ form incessantly, invading the entire finger, then other fingers and the hand, in islands or over a continuous surface. The other extremities are attacked in their turn. Radiating pains and pruritus are present. The affection lasts for years, with paroxysms, but without healing nor extending over the body. The nails finally fall out (compare impetiginous onyxis); the skin becomes atrophic and remains red.

Aside from this suppurative form of acrodermatitis, a vesicular form has been described, characterized by isolated vesicles, on a red base, incessantly reproduced on the same fingers and related to the zosteriform eruptions.

CHRONIC PEMPHIGUS.

This name, or that of genuine pemphigus (*pemphigus vulgaris*), is reserved by me, with Besnier and Brocq, for a rare and almost invariably fatal progressive bullous disease, the most dangerous among the great malignant skin diseases.

Symptoms.—Genuine pemphigus usually begins about the mouth, the pharynx or lips, the nasal fossæ [the conjunctivæ], or sometimes on the anterior aspect of the chest.

The eruptive lesion on the skin consists of round rather large-sized bullæ, of nummular dimensions, tense or flaccid, with yellowish or turbid contents. They develop very rapidly on the healthy skin; their base becomes reddened at the end of a few hours, or when they suppurate. Whether ruptured or not, they may undergo two different evolutions, either drying in crusts which are shed at the end of eight or ten days and leave a red or brownish macule; or the roof of the bulla becomes detached, exposing a smooth bright red surface which sometimes suppurates. They do not enlarge much, but multiply in incessant new crops.

In advanced stages of the disease the erosions are slow to become covered with epidermis; they run together here and there in polycyclic, raw or crusted lesions, surrounded by bullous elevations (Fig. 57) and may cover a considerable portion of the integument, more or less simulating eczema or an exfoliative dermatitis.

According to the contents and the behavior of the bullæ, the

terms *hemorrhagic, ulcerative, diphtheroid, gangrenous pemphigus,* etc., have been employed.

The eruption occupies especially the folds, the neck, the axillæ, the anogenital and inguinocrural regions, the umbilicus, the periphery of the nails, as well as points subjected to pressure, such as the buttocks, trochanters, scapulæ, knees, heels, ears; but there is a tendency to almost complete generalization.

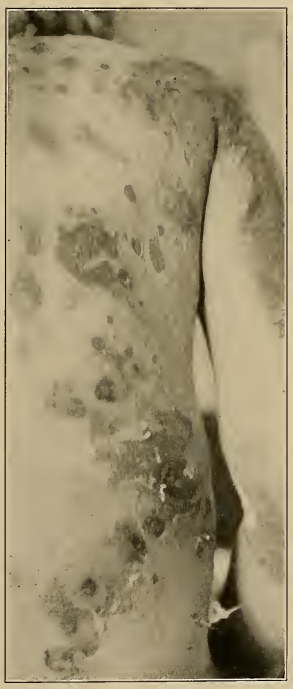


FIG. 57.—Genuine chronic pemphigus in an exhausted old man. The eruption dates a month back, the patient died less than seven weeks from the time of its onset.

In the mouth the lesions are primary or precocious, very transitorily bullous, assuming the appearance of a diphtheroid angina or ulcerative membranous stomatitis, and promptly spreading to the lips. The nasal, conjunctival, vulvar and other mucous membranes are very often affected in a similar way.

At a certain stage of the disease Nikolsky's sign is invariably present; by forcible pressure exerted on the patient's skin with the pulp of the finger, the horny layers can be detached and made to slip off; moreover, a moderate amount of pressure gives rise to the appearance of a bulla. This sign, which indicates acantholysis, is also met with in pemphigus foliaceus, in congenital pemphigus, and in the grave form of Duhring's disease.

Pruritus, formication and heat sensations may be entirely absent, the bullæ developing without the patient's knowledge when this is the case, in contradistinction to Duhring's disease. But the excoriations, sores and buccal lesions cause severe pain. Adhesions may develop between two mucous surfaces; serious changes of the conjunctivæ and corneæ may occur; the nails and hairs fall out.

FIG. 58.—Histology of genuine chronic pemphigus. Flaccid subcorneal bulla, very recently developed, derived from the case shown in Fig. 57. The cavity contains a sero-albuminous fluid in which float numerous white corpuscles and a few epithelial cells. The adhesion of the epidermal cells with each other is diminished; on the borders of the blebs, the corneal layer tends to become detached from the rete; Nikolsky's sign was present to a marked degree in this case. × 65.

The general phenomena consist of nervous depression or excitement, anorexia, extremely rapid emaciation; finally diarrhea, vomiting, cachexia and sometimes a transformation into pemphigus foliaceus, lead to death. Fever has been noted, being sometimes accounted for by the existence of abscesses, ulcers, or sloughs.

The course is rapidly progressive. The fatal termination takes place in three to eighteen months, rather less than more, through cachexia or an intercurrent infection.

Cases have been reported, and have come under my personal observation, possessing all the characteristics of true pemphigus, instead of Duhring's disease, which nevertheless terminated in recovery or in a complete remission lasting several years. We are obliged therefore to admit the existence of a benign form.

Diagnosis.—This is usually very precarious at the onset, and it has well been said that there exist no two exactly similar cases of

genuine pemphigus. The most experienced dermatologists sometimes hesitate in the presence of the early lesions which have the appearance of stomatitis, angina, toxidermia, hydroa, or Duhring's disease. In the developed stage, the clinical picture becomes clear and more or less approximates the description given above.

Pathological Anatomy.—The bullæ are so fragile that it is difficult to obtain unruptured specimens for examination. The cavity may be subcorneal, intramalpighian, or subepidermic. Underneath the bulla, the derma is edematous, but contains only a few wandering cells.

The subjoined figure (Fig. 58) gives an idea of these lesions.

Eosinophiles are not usually found either in the fluid contents of the bullæ or in the neighboring tissues. The contents are sterile or enclose microbes of secondary infections. Blood cultures are usually negative. The investigations of Lipschutz point to protozoa as the responsible agents. [?]

Etiology and Character.—Chronic pemphigus attacks especially weakened exhausted individuals past the age of forty years, somewhat more frequently males. The Jewish race seems to be predisposed to it.

The disease is not contagious, and there is nothing to prove its infectious character. It may equally well be claimed to result from a severe affection of the central nervous system, or possibly from an auto-intoxication.

Treatment.—The external treatment is that of the other bullous skin affections. Internal medication has for a long time been considered absolutely ineffective, so that the patient's strength was simply kept up by means of nourishing food, fresh air, tonic medicinal agents, strychnin, etc. Recently, various authors have reported improvement and even cures, by means of quinin (1.0 to 2.5 gm. daily), antipyrin, arsenic, and especially arsenobenzol, as well as by injections of normal serum. These therapeutic measures should accordingly be resorted to, instead of considering the situation as hopeless.

PEMPHIGUS FOLIACEUS.

This term is applied, since Cazenave, to a skin disease which begins with pemphigus and becomes transformed into an exfoliative erythroderma. It is difficult to give a clear description of its extremely variable clinical picture and course. The onset may be that of chronic pemphigus, or more rarely Duhring's disease; or there may be a bullous dermatosis impossible of classification, with rare and discrete, then numerous and close-set bullæ, often remarkably flaccid. Depending on its course, pemphigus foliaceus is called secondary or primary.

In the transition-stage, no more normal epidermis is formed at the points occupied by the bullæ, but instead lamellar, leaf-like, moist scales or crusts, which cover large red surfaces, sometimes extending over almost the entire body (Fig. 59). This epidermis is incapable of forming the roof of blisters and these are replaced by oozing spots from which the scales are easily detached. Under the scales or at the macerated points, a highly offensive epidermic mass

Fig. 59.—Primary pemphigus foliaceus of four years' standing, in a woman aged twenty-five years.

is found, made up of isolated Malpighian cells. On the borders of the squamous surfaces is seen a bullous margin, especially in the vicinity of the hands and feet which sometimes escape.

Finally, the lamellar exfoliation becomes general and resembles that of the other erythrodermias; it is sometimes so abundant that handfuls of epidermal structures can be picked up from the patient's bed. However, it usually retains a peculiar moisture.

The skin is a dark or brownish red, thinned and tense, or not infrequently papillomatous; fissures appear in the articular folds and ectropion develops on the eyelids. The hairs of the scalp and body become scanty; the nails become streaked and hooked and may fall out. The mucous membranes usually remain intact.

Pemphigus foliaceus is the disease in which Nikolsky described the sign of easy detachability of the horny layer, which he believed to be pathognomonic.

Pruritus and heat sensation are not prominent, except periodically. The urine is scanty and the urea and nitrogen index is always low.

The disease is protracted, sometimes lasting five to ten years, often only two or three years. It regularly terminates in death, due to digestive disturbances, especially diarrhea, marasmus, or intercurrent complications.

The *pathological anatomy* has yielded divergent findings. The epidermis was found to be tense and thinned or there were greatly elongated papillæ and intrapapillary buds, with dermic and epidermic edema and abundant exocytosis. The condition of the granular layer is variable. Eosinophilia is noted in the blood. Various lesions of the nerve centers have been described, but are inconstant.

Pemphigus foliaceus is encountered in both sexes, among adults in a state of physical or psychic deterioration.

We do not know if it is of nervous, toxemic, hematodermic, or other origin. It seems that certain cases can be interpreted as malignant exfoliative herpetides, secondary to a pemphigus; the appearance of other cases is that of a distinct pathological entity.

The *treatment* must follow that of the other bullous skin diseases and the erythrodermas.

CONGENITAL PEMPHIGUS.

This is a cutaneous malformation rather than a disease; it is very rare, usually familial or hereditary, as a rule congenital, but occasionally does not mainfest itself until late childhood, or still later. From various sources attention has been called to the apparent influence of consanguinity of the parents.

It consists of a predisposition of the skin and sometimes of the mucous membranes, to react in the form of bullæ toward all traumatisms, pressures, or even slight bruises.

Two degrees are known, and by some are considered as separate types of the disease: The first, *epidermolysis hereditaria bullosa* of Köbner, or *simple traumatic hereditary pemphigus,* consists merely of a tendency to the formation of tense, subcorneal, serous or sero-sanguinolent blisters; these develop in the healthy skin,

under the influence of blows, or pressure by the clothing, shoes, etc. The feet, hands, wrists, elbows, knees, etc., and even the buccal mucosa are affected with blebs, which come on without pain in less than an hour after the traumatism and which heal very readily, provided infection does not occur.

In the second, the grave and dystrophic form—"*Pemphigus successif à Kystes épidermiques*" or *congenital pemphigus with a tendency to cicatrization*—an apparently spontaneous production of more or less numerous bullæ is noted at various points of the limbs and body, within a short time after birth. There is a constant succession of these bullæ, especially in regions exposed to pressure, on the extremities, the ears, the knees and elbows, even on the mucous membranes.

Gradually the skin of certain regions, principally that on the back of the hands and the joints of the fingers, the elbows and knees, etc., becomes atrophic or cicatricial, thin like onion peel, of a brownish purplish-red color. An enormous number of very small white opaque granules are seen; these are milium cysts. The nails undergo various deformities, or fall out permanently.

The microscope discloses atrophy of the derma with disappearance of the papillary body and shows that the cysts are dilatations of the sweat ducts with corneal contents or more rarely follicular cysts. Similar miliary cysts and atrophic changes are noted in some cases of Duhring's diseases.

In the congenital forms of pemphigus, Nikolsky's sign of easy detachability of the horny layer is almost invariable present. In certain cases, a combination has been demonstrated between congenital pemphigus and various forms of congenital ichthyosiform hyperkeratosis. On the other hand, Nicolas, Montot and Charlet have described a type with chronic and progressive vegetative ulcerations.

The pathological predisposition usually diminishes with the advance of years; or it may become limited to some particular region.

The patient must be cautioned against all exposure to traumatism. Protective dressings and tonic internal medication are indicated.

PEMPHIGUS HYSTERICUS.

Hysterical pemphigus does not exist, as has been shown by investigations and conclusive discussions.

Under this name, or under that of *virginal* or *chlorotic pemphigus*, were designated attacks of bullous eruptions, hemorrhagic, pustular or sloughing, which occurred in nervous girls or young women of peculiar character.

The lesions, often of an irregular or elongated shape, are distributed in a remarkable fashion, sometimes with suspicious regularity. They appear one by one, or in small numbers at a time, throughout a period of several months or years. The course is invariably benign. However, through their extent, numbers and sometimes gangrenous character, the lesions may cause actual mutilations.

In all cases which could be seriously investigated, where the patient was carefully watched, or when, for example, a sealed occlusive dressing was applied over the affected region, the condition could always be traced to malingering. The lesions are burns or cauterizations by chemical or medicinal agents and are produced by the patient herself under the influence of a special mental state which has been named *mythomania* or *pathomania*. These are cases of *dermatitis artefacta*. The truth of Babinski's statement is beyond question, that hysteria is incapable of giving rise to trophic disturbances of the skin.

The exposure of trickery demands some tact on the part of the physician, whose suspicions are often repudiated by the patient's environment. But when the proof of the fraud has once been made, the so-called eruption subsides of its own accord.

CHAPTER XI.

KERATOSES.

HYPERKERATOSES AND DYSKERATOSES.

The name keratosis is applied to a dermatological lesion which consists in moderate thickening of the horny layer; hyperkeratosis means a considerable hyperplasia of this layer; dyskeratosis is a pathological process in which a disturbed keratinization terminates in the formation of an abnormally constituted corneal layer, with thickening in some of the cases and disintegration in others.

Under normal conditions, the horny layer is formed by the superposed layers of lamellar cells, which are composed of keratin and loaded with fat, but have neither protoplasm nor nuclei. These corneal cells represent the ultimate stage of the process of epidermic development. Originating through multiplication of the constituents of the basal layer, the Malpighian cells are gradually pushed up by the newly generated cells and reach the granular layer where they take up eleidin or keratohyalin, after which they suddenly undergo the transformation known as keratinization.

The corneal cells adhere to each other and persist a certain time at the epidermal surface, where they constitute a resistant, supple, and only slightly permeable protective covering. Being no longer alive, the horny cells are incapable of vital reaction against irritants of any kind and are finally shed.

The thickness of the normal horny layer varies slightly in different individuals and greatly in different regions of the body.

I shall begin with a discussion of the cutaneous affections characterized by thickening of the horny layer, leaving to the end of the chapter the conditions designated dyskeratoses.

KERATOSES AND HYPERKERATOSES.

In the cutaneous affections characterized by a thickening of the horny layer the degree of this hyperplasia is variable—being slightly marked and accompanied by a powdery or scurvy desquamation in *kerosis* and *pityriasis simplex*—or considerable, often producing a genuine carapace, resistant and liable to crack, in the ichthyosiform hyperkeratoses and numerous keratodermias; in *ichthyosis*, all degrees are met with, from simple xerodermia to the most pronounced sauriasis.

In a general way, when the horny layer is very hyperplastic, the granular layer and the mucous body are, as a rule, likewise of very abnormal thickness; the derma itself is usually congested and there is an obvious tendency toward the production of papillary elevations or vegetations.

The name of *verrucose state* is applied to the common combination of keratosis with the vegetative process (XII).

The term *keratoma* is sometimes applied to a circumscribed hypertrophy, forming a tumor, such as cutaneous horns.

From the point of view of the distribution of the lesions on the surface of the integument, a distinction must be made between the following groups:

1. *Diffuse and generalized keratoses*, spreading over almost the entire body, or at least over large surfaces, although with evident *regional* predilections.

In this group I shall describe *kerosis, pityriasis simplex, ichthyosis* and the *generalized and partial ichthyosiform hyperkeratoses.*

2. *Circumscribed keratoses*, composed of distinctly limited keratotic spots or surfaces; some are scattered without apparent order, others assume a regional or symmetrical arrangement, while a few are even distinctly systematized.

This group comprises: *keratotic nevi, linear nevi, senile warts, senile keratosis* and a few analogous affections.

It is not superfluous to point out that keratotic lesions in scattered spots or on more or less extensive surfaces, may be present or simulated, in a large number of skin affections. But corns, flat warts and common warts have been described elsewhere. The cutaneous dystrophies such as acanthosis nigricans, which is vegetative and xeroderma pigmentosum, in which the lesions of the derma are important and essential, likewise belong in other chapters.

As regards the patches of psoriasis inveterata and ostreacea, lichen hypertrophicus, lupus erythematodes of the type of "herpes cretacé," tuberculosis verrucosa, the angiokeratomas, etc.—the thickening of the horny layer here is secondary to a definite process of different character. They are here mentioned only from the viewpoint of the differential diagnosis.

3. *Regional keratoses* proper, which owe very peculiar characteristics to their topographical localization. Such are notably the palmar and plantar keratoses, for which I reserve the name of *keratoderma.*

Although the epithelium of the mucous membranes, more particularly that of the mouth, does not, under normal conditions, become keratinized like the epidermis, local pathological lesions are observed which are properly entitled to the name of *keratoses of the mucosæ.*

KEROSIS.

The chronic pathological condition of the skin which I have named kerosis, is characterized clinically by: (1) Dirty yellowish or grayish coloration; (2) accentuation of the pilo-sebaceous pores; (3) a slight thickening of the integument.

The anatomical lesions are: A slight diffuse hypertrophy of the horny layer, with a tendency to fine desquamation and a modification, of unknown character, of its fat content; hyperkeratosis of the pilo-sebaceous orifices.

This dystrophy finds its proper place in the group of the diffuse keratoses. It is usually misinterpreted and confused, erroneously in my opinion, with *seborrhea*, which constitutes merely a complication.

Although of slight importance in itself, kerosis acquires clinical interest because it furnishes the necessary or ordinary substratum for several of the most common cutaneous affections. Such are certain *pityriases*, *seborrhea*, certain *alopecias* and *hypertrichoses*, *hyperidrosis oleosa*, some *acnes*, *rosacea*, many *eczematides*.

The kerotic pityriasis will be described in the following paragraph. For the other kerotic affections, the reader is referred to the chapters dealing with the folliculoses, trichoses, hidroses and the erythematosquamous dermatoses.

The topographical distribution of kerosis is both diffuse and regional. It occupies by predilection and to the highest degree, the center of the face, especially the nose and the nasogenial furrows, and the scalp; very frequently also the forehead, the temples, the chin, the nape of the neck; on the trunk, it affects especially the pre-sternal oval and the interscapular groove, spreading more or less around the middle line. It often covers the shoulders, the entire thorax, uniting at the sacrum behind, at the umbilicus in front; it is not uncommon on the pubis, the genital organs, the intergluteal fold, in the large articular folds and even on the palms of the hands.

The following parts invariably escape: the front of the neck, the extensor surface of the limbs, the buttocks and the forearms and legs as a whole.

It will be noted that this distribution is practically the opposite of what is seen in ichthyosis.

On this territory, the various manifestations and complications of kerosis are not produced haphazard, but each has its preferred or exclusive regions.

Numerous factors play a role in the *etiology* of kerosis. This anomaly is so widely distributed in civilized countries and in its mild degrees so close to the physiological condition, that one hesitates to define it as a disease. Certain individuals, more or less

numerous according to the race, are immune. Direct heredity, or the influence of poor health or bad hygiene of the parents, constitute the fundamental conditions of kerosis. The establishment of the genital functions and sexual disturbances on the one hand, unhygienic diet on the other, in the form of a hypernitrogenous diet, abuse of stimulants, coarse food, insufficient mastication, abnormal fermentations, constipation, etc., are common factors, the individual effect of which is not easily determined.

External local irritation, reflex circulatory disturbances connected with affections of the mucous membranes, play a much less prominent part.

By a certain school, the manifestations of kerosis are interpreted as directly microbic, each one, according to Sabouraud, being referable to a special variety of microbe or an association of varieties. The presence of microörganisms is indeed undeniable, but their pathogenic action has not been demonstrated.

At any rate, kerosis and its manifestations follow a certain law of *evolution* in connection with the age of the individual. Dry pityriasis of the hairy scalp, appearing at the age of six to ten years, becomes transformed about or after puberty into oily pityriasis, seborrhea developing at the same time. Acne juvenilis flourishes from the age of fifteen to twenty-five years. Calvities gravis begins at twenty-five years, or less. Rosacea may be premature, or be delayed until about the age of forty-five years. In aged individuals, the sequelæ of kerosis become attenuated and extinct, unless it be held accountable for seborrheal warts and senile keratosis.

It is quite possible that the vernix caseosa, or the greasy epidermic covering of some infants at birth and the so-called acne miliaris of the newborn which may accompany it, represent the first manifestation of kerosis. This would be an additional reason for regarding the latter, accepting the views of Jacquet, as closely related to sexual development. As a matter of fact, kerosis has two periods of florescence, that of the genital wave at the time of birth, and that of puberty; and a period of decline, when sexual life is restricted.

The *treatment* of kerosis must consist in the first place in correcting all hygienic deficiencies which may be present. It may be necessary to administer alteratives, such as phosphates, cod-liver oil and especially arsenic on account of its keratoplastic properties.

Mineral springs, with sulphur, arsenic or sodium chloride water, are especially indicated.

Locally, sulphur in the form of sulphurous or sulphur lotions, soaps, glycerolates, pastes or salves, but also the tars, camphor, calomel, reducing agents in general, are very serviceable.

With perseverance, it will almost invariably prove possible to obliterate the principal kerotic manifestations and to restore the skin, if not to normal, at least to a condition very close to normal; relapses, however, are common.

PITYRIASIS SIMPLEX.

The name of pityriasis simplex is applied to a non-inflammatory, scurfy, bran-like desquamation of the horny layer.

The term pityriasis, derived from the Greek πίτυρον meaning "bran," is on the other hand applied to various pathological conditions which have nothing in common with the subject under discussion.

Pityriasis versicolor is a specific parasitical disease of the epidermis; *pityriasis rosea* of Gibert is an erythemato-squamous dermatosis; *pityriasis rubra pilaris* belongs rather with the folliculoses; *pityriasis rubra* is an erythroderma.

Pityriasis simplex must be distinguished from the pityriasiform desquamations following upon an inflammation, such as may be observed after the erythemas, eruptive fevers, pyodermatitides, or in the course of certain trichophytoses and especially of eczemas. In these cases, the desquamation almost invariably results from the phenomenon known as parakeratosis.

In pityriasis simplex, on the contrary, the keratinization of the epidermis is complete, and takes place, at least apparently, in a normal manner; but the corneal epithelium is thickened and peels off in scurfy lamellæ or scales, instead of in a fine powdery and imperceptible form as on the healthy skin.

In accordance with this definition, pityriasis simplex is one of the most common manifestations or consequences of kerosis. Hence there is nothing to add in regard to its etiology, to what has been stated concerning this dystrophy; but I wish to emphasize again the influence of age upon its course.

Those authors who include the entire picture of keratosis under the incorrect name of "seborrhea," have been led, after Hebra, to consider pityriasis simplex as a "*seborrhea sicca;*" but this expression is in every way inadmissible.

Pityriasis simplex is almost exclusively met with, or at any rate decidedly predominates in the hairy regions and especially on the scalp, where it is designated as *pityriasis capitis*. Next in order, it affects the beard, the pubis, the hairy regions of the thorax and sometimes the limbs.

Two varieties can be distinguished, which are connected, however, by a series of intermediary forms.

Pityriasis sicca is a condition in which there is an incessant reproduction of dry white or grayish lamellæ, called dandruff by the laity. An extreme degree was formerly described as "teigne amiantacée (asbestos-like tinea), a name suggested by Alibert. Its mild degrees blend with the physiological desquamation which is encountered on any neglected scalp or improperly kept beard and even on the entire body of bedridden patients.

In *pityriasis oleosa*, which often follows upon the preceding, the scales are oily, greasy, yellowish, sticky; but it is noteworthy that they occur on a skin of normal coloration, without pathological redness.

The oily character of this pityriasis might appear to be referable to its association with seborrhea (supra-seborrheal pityriasis of Sabouraud). This combination is frequent, but not constant. There is an oily pityriasis without seborrhea in the same location; the fat is derived from the keratinization itself.

As regards the steatoid pityriasis of Sabouraud, this is not a simple pityriasis, but an eczematide on a kerotic soil. It furnishes no scales, but crusts, the consistence of which is due to dried serum; the skin where they occur is pink and moist and pruritus is often present. The so-called milk-crusts of children usually belong to this variety.

Although we are not justified, in my opinion, in regarding pityriasis simplex as of parasitical origin, it is necessary to point out the remarkable abundance of microörganisms in its scales. The predominating parasite is the spore of Malassez, or bottle bacillus; it is very polymorphous, rather large, assumes the forms of yeasts, but has so far resisted all attempts at artificial culture. It is associated with the bacillus of seborrhea in oily pityriasis, and with various cocci, chiefly the polymorphous coccus of the skin (*coccus cutis communis*) in the so-called steatoid pityriasis.

Pityriasis simplex is merely an unpleasant, hardly a troublesome affection, in itself; it is feared only on account of the alopecia and baldness which are attributed to it. Aside from this, it exposes to the danger of eczematides and must therefore be systematically controlled.

The *treatment* of pityriasis capitis requires in the first place local measures; frequent washing with sulphur, naphthol or tar-soaps, or better with a decoction of quillaya bark [saponin], aqueous solutions with sulphur, coal tar, etc., or alcoholic and ethereal, mercurial, naphtholated or compound lotions, several formulæ for which may be found at the end of this book. Ointments are not agreeable to the patient, but it is advisable to employ them in severe cases, at least for nocturnal treatment.

The general hygiene and that of the hair and its arrangement

must be properly regulated. Analogous measures are applicable in pityriasis of the beard or other regions.

Pityriasis Simplex of the Face and Hairless Parts is limited to children and youthful individuals with delicate skins. It is found around the mouth, on the cheeks, the chin, the front of the neck, and sometimes on the trunk and the limbs. It appears in the form of rounded, oval spots or more or less distinctly outlined polycyclic, scurfy or furfuraceous areas, with a normally colored or slightly pinkish surface, lighter than the background when the complexion is tanned or sun-burnt.

This superficial dermatosis is contagious and even epidemic in groups of children. It may coincide with impetigo and Sabouraud regards it as a dry impetiginous epidermatitis, due to staphylococci. It may become eczematized and should probably be classified with the mild eczematides.

It is very readily curable by means of mild sulphur or white precipitate ointments, etc.

ICHTHYOSIS.

Ichthyosis is a diffuse generalized keratosis which is never congenital but which manifests itself at an early age and persists throughout life. It is usually considered a malformation of the skin.

Symptoms.—The ichthyotic skin is dry and scaly. In typical cases of moderate severity, the integument is roughened, parchment-like, covered with dry scales which have been compared with fish scales; they are thin, white or brownish or gray, more or less easily detached and constantly renewed.

Many degrees or objective varieties of ichthyosis can be described: In xeroderma the skin is merely dry and the desquamation is powdery, almost imperceptible; "ichthyosis nitida, or nacreous ichthyosis, with thin and silvery lamellæ, is the most common form; in ichthyosis nigricans or black ichthyosis, the scales are of a dark color; they are large and polygonal in ichthyosis serpentina; large, thick and resembling the skin of the crocodile, in sauriasis; finally, in ichthyosis hystrix, prominent warty or pointed horny excrescences are seen, suggestive of the porcupine's skin.

The two last named varieties are sometimes combined under the name of *ichthyosis cornea;* it is very probable that they do not legitimately belong to ichthyosis but to the ichthyosiform hyperkeratoses, which will be discussed further on.

Ichthyosis is always symmetrical and is most marked on the extensor surface of the limbs, especially the elbows and knees (Fig. 60), but also the trunk and to a less degree, the head and hands and

feet. The face is, as a rule, only slightly xerodermic; the scalp is pityriasic; the palms of the hands and the soles of the feet are very often dry and wrinkled like the hands of washerwomen.

On the other hand, the articular folds, axillæ, elbow bends, popliteal spaces, intergluteal fold, groins and the genital organs are always more or less free, in contradistinction to what is seen in the ichthyosiform hyperkeratoses. Lesions of mucous membranes are altogether absent.

On passing the finger-nail or some pointed object over the skin of ichthyotic patients, a white powdery track is left behind; the skin often appears tense and when it is grasped between two fingers the papillary body folds up superficially over the derma. The hairy system may be normal or is imperfectly developed; the downy hairs of the extensor surfaces are very fine, resembling lanugo and sometimes appear to be thinned; the co-existence of keratosis pilaris is invariable.

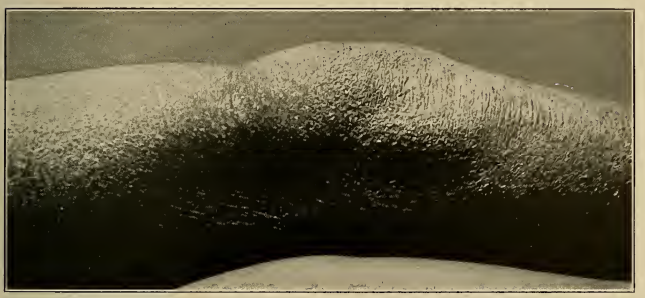


FIG. 60.—Ichthyosis nitida in a youth aged sixteen years. Note the white mark left by the garters above the knees.

The nails are normal, or rarely dry and brittle. The sebaceous and sweat secretions are greatly diminished; heat or violent exercise may cause sweating in mild cases of ichthyosis and the affection will then become attenuated or disappear, as for instance, in the summer. Ichthyotic patients are usually thin, poorly developed and of slight resistance.

Pruritus is not a feature in the clinical picture of ichthyosis, unless it is complicated by *eczema*, which is rather common. This eczematization is observed especially in persons neglectful of cleanliness and those exposed to artificial dermatitis, or even, it has been claimed, without an apparent local cause; the attacks may be obstinate and recurrent but always represent merely a superadded complication.

Ichthyosis is never strictly congenital; it develops gradually and is usually not observed until the third year of life, though sometimes as early as the third or fourth month; often the date of onset cannot be accurately determined. It may become attenuated at the time of puberty, but as a rule it persists until death. Hebra and Hardy assert that it may disappear after eruptive fevers, but this is extremely doubtful.

The name of *ichthyosis tabescentium*, desquamation of cachectics, and *senile ichthyosis* has been applied to a state of diffuse atrophy, with dryness of the skin and ichthyosiform desquamation, which develops in weak old people and bedridden invalids.

It is not known if the pathological condition in these cases is always identical and how it is related to ordinary ichthyosis.

Pathological Anatomy.—The horny layer is always more or less thickened; the granular layer is diminished or absent in the variety *nitida*, but there is no complete parakeratosis; the mucous body is rather thin and tense. The papillæ are less developed than normal. The papillary body and the chorion are almost invariably found to contain a moderate infiltration of round cells and mast cells around the vessels; this fact has led Unna and Tommasoli to consider ichthyosis as an inflammatory skin disease. The pilosebaceous follicles present the lesions of keratosis pilaris. Various lesions of the sweat glands have been reported.

The changes attributed to the *hystrix* form are identical with those of ichthyosiform hyperkeratosis and of the hyperkeratotic nevi; so that some doubt prevails as to the existence of a true ichthyosis hystrix.

Etiology.—Ichthyosis, except in its mildest form, is not very common; its extreme degrees are actually rare. It is a hereditary malformation in one-fourth of the cases, familial in one-half of the cases, according to Gassmann; the transmission is irregular and may skip a generation. It is not certain and even rather improbable that alcoholism, syphilis or tuberculosis of the parents create a predisposition to the disease. Both sexes are equally affected.

Diagnosis.—Mild xeroderma with or without keratosis pilaris, may be misinterpreted or confused on superficial examination with pityriasis simplex, kerosis, the post-eruptive desquamations, desquamations of cachectics, etc. When the onset of the affection can be determined, the point will be settled at once.

In psoriasis, in the dry eczemas and in pityriasis pilaris, the skin is red. Psorospermosis, named ichthyosis follicularis by J. C. White, is quite characteristic.

Ichthyosis must be differentiated especially from *generalized* ichthyosiform hyperkeratosis (congenital ichthyosis of certain

authors), and from regional or very extensive systematized *hyperkeratotic nevi*, many cases of which have been described under the names of ichthyosis partialis, ichthyosis hystrix, etc.

Treatment.—Although the disease is incurable, the patients can be greatly relieved by maintaining their skin in a clean and supple condition. Arsenic seems to be of little use; cod-liver oil is to be recommended; thyroid medication may be cautiously tried.

The external treatment, however, is of the greatest importance. Repeated and prolonged baths, washing with soap, steam baths, on the one hand and daily inunctions with vaseline, glycerin, fatty substances or various salves, on the other, will give the skin in a short time a nearly normal appearance in cases of moderately severe ichthyosis. The same measures must be insisted upon in the severe types. It is necessary to regulate the treatment in a given case in such a way as to keep up the results attained.

ICHTHYOSIFORM HYPERKERATOSES.

Under this heading must be grouped some interrelated dermatoses which differ from ichthyosis by the following characteristics:

They may be strictly congenital or their appearance may be delayed for a shorter or longer time after birth; they usually take a progressive course; hyperkeratosis is more pronounced than is ichthyosis and is accompanied by a marked redness of the skin which has led to the name of *ichthyosiform erythroderma;* anidrosis is absent or may be actually replaced by hyperidrosis; far from sparing the articular folds, the lesions on the contrary are especially well developed in these regions; their histological structure is altogether different.

A distinction is made between a generalized form, to be described next, and circumscribed forms, which will be discussed in the succeeding paragraphs.

Generalized Ichthyosiform Hyperkeratosis.—This is often named fetal, intra-uterine or congenital ichthyosis, for it is always present at the time of birth. Two degrees of the affection may be distinguished.

The severe type is incompatible with life; it has been referred to in the chapter on the erythrodermias. It represents a diffuse congenital malignant keratoma.

The benign type, distinctly separated from ichthyosis by Unna under the name of *congenital hyperkeratosis*, is characterized by a more or less severe and universal redness of the skin which is contracted and covered with large thick, polygonal, brownish scales resembling the scales of sauriasis, very adherent, but capable of being detached in a single piece by traction or maceration (Fig. 61).

The face is affected; it is pinkish and scaly; ectropion is usually present. The articular folds are the seat of horny, blackish, papillary excrescences. The palmar and plantar regions present the appearance of hereditary keratoderma. The hairy scalp is covered with a sebaceous coat. Brocq observed that in certain

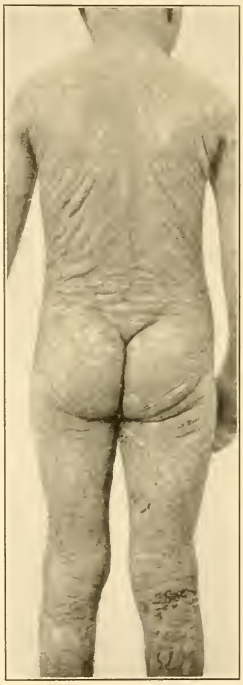


Fig. 61.—Generalized ichthyosiform hyperkeratosis in a girl aged eight years. Note the lizard-like aspect of the integument and the horny vegetations in the popliteal spaces.

cases, which he calls *ichthyosiform congenital erythroderma with hyper-epidermatrophy,* the hairs and nails grow two or three times as rapidly as in normal individuals.

Especially in the first years, occasional crops of bullæ may be observed on the limbs and on the trunk; I have shown that these bullæ are auto-inoculable; they probably represent merely an

impetigo, favored by the cracking of the epidermis (Fig. 62). All the symptoms, especially the redness, become attenuated with age.

The majority of cases described under the names of *ichthyosis cornea, sauriasis, ichthyosis hystrix*, were in reality ichthyosiform hyperkeratosis.

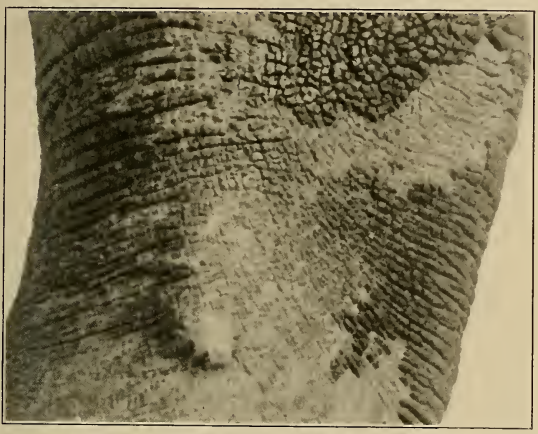


FIG. 62.—Generalized ichthyosiform hyperkeratosis in a boy aged thirteen years. External surface of right knee. The horny verrucous covering has been detached in places; at the level of the head of the fibula a bulla is seen, resulting from an experimental auto-inoculation.

The *pathological anatomy* shows lesions altogether different from those of ichthyosis. The horny layer is enormously thickened and arranged like shingles on the acuminate protuberances; it contains considerably less fat than in the normal condition. The granular layer is markedly hypertrophied. The rete is thickened. The papillæ are greatly elongated and irregular. Perivascular infiltration is not constant.

Partial Ichthyosiform Hyperkeratoses.—Clinical observation and histology are in accord as to the relation with ichthyosiform hyperkeratosis, of familial keratoderma or Méléda disease, which seems to represent merely a regional and partial variety of this affection (p. 211).

Notwithstanding its very remarkable course, it seems justifiable to connect with ichthyosiform hyperkeratosis also the rare disease symmetrical erythro-keratoderma, especially since it may become associated with familial keratoderma (Brocq and Dubreuilh).

This uncommon skin affection, a typical case of which was reported by me in 1911, the face and upper part of the trunk alone escaping, appears after birth in the form of spots or isolated patches which spread rather rapidly and finally invade almost the entire integument; the keratotic surfaces may become warty and give rise to genuine cutaneous horns.

All the hyperkeratoses of this group, generalized or circumscribed, probably represent cutaneous malformations of the same kind as nevi; their sometimes delayed appearance, their symmetry and their extensive character, do not militate against this view. There are reasons for the belief that consanguinity of the parents and heredity play a certain part in their etiology.

CIRCUMSCRIBED KERATOSES.

Keratotic Nevi.—As with nevi in general, these may exist at birth, or develop in the course of childhood or even later. They appear in the form of spots, elevations or verrucosities which gradually become covered with a more or less thick coating of horny tissue.

Hyperkeratotic and Varicose Nevi.—Hyperkeratotic and varicose nevi may be single or multiple, limited or very extensive, situated at any point of the integument. Some are *regional* and occasionally *symmetrical*, occupying for example certain articular folds. Others are covered with such a thick accumulation of horny substance as to constitute a genuine *cutaneous horn*, resembling a small ram's horn.

Linear Nevi.—Linear nevi constitute a very peculiar systematized form of hyperkeratotic nevi. The verrucosities, of a gray, brown or black color, are arranged in continuous or interrupted streaks of great length and variable width, often in very regular patterns. Sometimes unilateral, in other cases symmetrical, these nevi are made up by a single streak or several streaks following a parallel course. I have repeatedly observed the entire body, including the limbs and the face, to be covered by linear designs of this kind (Fig. 63). On the limbs the streaks are longitudinal, often partially spiral. On the trunk, they are horizontal or rather oblique and frequently present angular inflections, forward and backward, in the vicinity of the middle line, which itself may be traced by a streak. In the face, the lines are orbicular or radiating in various directions.

An explanation of these distributions which do not seem to be the result of accident, has taxed the ingenuity of observers. The direction of the streaks has been referred to that of the nerves; to the lines of Voigt, separating the nerve territories; to the lines of cleavage of the skin; to the course of the bloodvessels; to the metameric

zones of Head; to the lines of fusion of the embryonic clefts; finally, to stretching of cell-groups in the course of embryonic growth.

None of these theories explains all the arrangements which have been noted; and the terms of nervous, zoniform, metameric, unilateral, etc., nevi, are not justified. The same remark applies to the term ichthyosis hystrix.

Fig. 63.—Linear hyperkeratotic nevi, very numerous, in a girl aged seven years. Service of Dr. Variot.

The linear nevi, instead of being hyperkeratotic, may suggest simple papilloma, psoriasis, or lichen; they may be hairy, pigmentary and so forth.

Not all linear dermatoses are necessarily nevi; in rare instances, genuine cases of lichen planus, psoriasis, eczematides, prurigo

vulgaris, etc., are observed which assume a topography like the linear nevi, appearing at any age and subsiding on appropriate treatment.

Flat Senile Warts (Verruca Plana Senilis).—These keratotic elevations, also known as *seborrheal warts*, have the following characteristics:

They are rounded or oval, sometimes irregular, from the size of a lentil to that of a green almond, more prominent in the center than at the periphery, distinctly circumscribed, sometimes actually overhanging, covered with an adherent horny and fatty layer, of variable thickness, of a gray, brown or black color. After this coat has been removed by washing with soap, maceration, or rubbing with ether, a mammillated, honeycomb or cauliflower-like surface with furrows is exposed; the consistence is molluscoid or granular.

Senile warts, generally numerous on the same individual, preferably occupy the flanks, the belt-line, the back, the chest, the neck, the shoulders; many hundreds of them may be counted; they are less frequently observed on the forehead, the temples and the cheeks. They develop after the fortieth year of life, especially in women; they may begin before the age of thirty. They persist and multiply with the advance of years.

Histology shows either hypertrophy of the epidermis or atrophy through invasion of the horny layer, but always some irregularity; the papillæ are deformed and twisted. Horny pearls are often found in the interpapillary depressions. A special lesion, described by Pollitzer, consists in a peculiar arrangement in whorls of the Malpighian cells at certain parts. The hairs and glands are atrophied; there is no true seborrhea. The subjacent cutis is often in a state of senile degeneration, but presents no inflammatory infiltration.

Their clinical features, their structure and their seat, everything *differentiates* senile warts from the patches of senile keratosis with which they are too often confused. They have not the same tendency as the latter to undergo epitheliomatous transformation.

I consider them as delayed nevi, and they actually coincide frequently with vascular nevi, fibroma molluscum, or pigmentary spots. There is no connection as regards etiology and character between senile warts and ordinary warts.

Treatment is instituted only on special request; mercurial ointments, collodium with salicylates or sublimate, black wash and even strong caustic agents may be recommended. It is much more advisable, however, to resort to the galvanocautery cautiously handled and combined if necessary with curettage; no perceptible cicatrix must be produced. Radiotherapy seemed to me to be inefficient.

Electrolysis is very successful, but the method is much too laborious in its application. [Destruction by means of carbonic acid snow may be employed.]

Keratosis Senilis.—The usually multiple and scattered keratotic spots which are observed especially on the face of aged individuals, were formerly known under the names of *"crasse des vieillards" acné sebacée*, etc. They seem to me to constitute a complication of senile degeneration of the skin and frequently lead to multiple epitheliomatosis (p. 685); from this viewpoint, they constitute an obvious type of precancerous dermatosis.

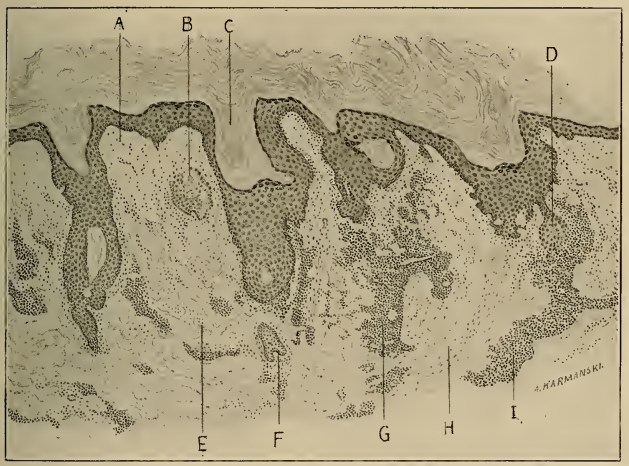


FIG. 64.—Histology of senile keratosis. The section comprises the border of a fairly extensive keratotic spot from the temporal region of an old man. *A*, edema; *B*, epidermic globe; *C*, hyperkeratosis; *D*, sebaceous gland; *E*, elacin; *F*, hair follicle; *G*, plasmatic infiltrate; *H*, elacin; *I*, plasmatic infiltrate. × 37.

The lesions of senile keratosis begin as dry yellowish or brownish spots, or as warty elevations somewhat resembling seborrheal warts, or again as irregular but fairly well outlined telangiectatic red spots. Gradually they become covered with a gray or brownish keratotic coat, friable or of dry consistence, with a wrinkled surface which may bristle with irregular elevations. This very adherent keratotic layer sends conical processes into the derma; its detachment often gives rise to small hemorrhages; the changes become less marked toward the borders of the spots; the skin in their center may be atrophic or cicatricial.

Senile keratoses appear more or less soon after the fiftieth year in very variable numbers, notably on the forehead, temples, nose, cheeks, the back of the hands and wrists, sometimes on the neck and the forearms. As a rule, they persist indefinitely and increase in number, but they may disappear.

Their evolution into epithelioma is by no means necessarily fatal. This is indicated by a transformation of the keratotic layer into a crust, ulceration of the subjacent derma, at first superficial, and the appearance of epithelliomatous pearls at the circumference.

Histology shows in the first stages an irregular hyperkeratotic horny layer, provided with conical processes on its inferior surface; a thinned mucous body, sometimes infiltrated with wandering cells; an irregular and edematous papillary body. The corium presents to a high degree the lesions of *senile dystrophy*, namely the transformation of the elastic tissue into basophilic elacin, and a colloid change of the connective-tissue strands; perivascular tracks of cellular infiltration are seen, with a predominance of plasmocytes (Fig. 64).

The *differential diagnosis* must be made from the various nevi, syphilides, psoriasis, rosacea, especially from lupus erythermatodes. The patient's age and the location of the lesions are factors of prime importance.

Treatment with salves and ointments containing keratolytic and keratoplastic medicinal agents is often not particularly efficient. Radiotherapy accomplishes frequently, though not invariably, a complete disappearance without pain. The galvano-cautery, assisted by curettage, yields excellent results.

In *pre-senile dystrophy*, in *xeroderma pigmentosum*, in *radiodermatitis*, and in *cutaneous arsenic poisoning*, practically identical keratoses in spots and warty elevations may occur; these dystrophies belong to the same natural group.

Gonococcal Keratoma.—A very rare variety of disseminated keratosis has been described under the name of "blennorrhagic keratoma" by Vidal, Jacquet, Jeanselme, Chauffard, and others. Certain cases of gonorrhea, confined to bed on account of severe arthropathies or myelopathies, present hard yellowish conical elevations, comparable to drops of yellow wax or upholsterer's tacks, scattered over the limbs and sometimes on the trunk. There may be an erythematous border at the circumference.

Genuine carapaces of hyperkeratosis have been observed in the palmar and plantar regions. These products disappear in two or three months under simple attention to cleanliness.

Porokeratosis.—The porokeratosis of Mibelli (1893) and Respighi is a rare affection, characterized by irregular circinate spots, the area of which is atrophic, scaly or normal, with a papular border

marked by a horny plate; this plate is imbedded in a "trench," or groove from which it rises in the form of a prismatic crest. The lesion begins with a horny cone wedged in a papule which slowly spreads out. The lesions are situated especially on the extremities, the face and the genitals; they have been observed in the buccal mucosa.

Porakeratosis is often familial, but nothing more is known concerning its etiology. Truffi associates it with the nevi. Mibelli assumes a primary epidermal dystrophy of the glandular orifices, more particularly of the sweat-pores.

The porokeratosis of Italian authors must not be confused with the *punctiform keratosis* which will be briefly referred to in the following and which the majority of writers also call porokeratosis.

KERATODERMA.

I employ the term keratoderma for the palmar and plantar keratoses.

The skin of the palmar and plantar regions has a special structure. It is predisposed to hyperkeratosis and in this case has a tendency to crack, forming very painful fissures in the flexion folds. All dry dermatoses in this locality assume an analogous appearance, which renders the diagnosis rather difficult.

Some keratodermas are *essential*, representing malformations; others are *symptomatic*, the result of repeated traumatisms, intoxications or localization of various dermatoses.

Essential Keratodermas.—**Familial keratoderma**, or **Méléda disease** [keratoma palmare et plantare] is an ordinarily congenital, sometimes acquired affection, which frequently attacks several children of the same family and has a marked tendency to hereditary transmission.

The palmar aspect of the hands and fingers (Fig. 65), the plantar aspect of the feet and toes, are symmetrically and as a whole, the seat of a horny thickening. The borders are marked by a purplish or bluish-pink edge from 4 to 5 mm. wide.

The hyperplastic horny layer may be smooth, soft, of a waxy or brownish-yellow hue, made up of large very adherent lamellæ. In this case a local hyperidrosis is frequently noted which is regarded by Lenglet as the initial phenomenon and the hyperkeratosis itself is supposed to originate at the sweat orifices. I have sometimes noted the presence of intracorneal bullæ with turbid contents.

In other cases, the hyperkeratosis is dry, hard, roughened, of a thickness which may reach a centimeter; it is fissured in the folds, or divided into polygonal blocks.

The underlying skin is invariably red, usually tense, sclerotic

and atrophic. In well-marked forms, it is so much retracted at the last phalanges that the fingers are conical and tapering, as if enclosed in a very tight-fitting, yellowish and horny case; the nails are thinned and the nail bed is bloodless.

The movements of the hands and fingers are impeded and painful; walking is difficult.

The lesions almost invariably encroach upon the anterior aspect of the wrists and along the Achilles tendons; the knees, elbows and articular folds may present thick, warty, *aberrant patches*, of a brownish-red color, distinctly circumscribed, with dilated and blackened pores. These patches may progress and extend, as shown by casts preserved in the Museum of the St. Louis Hospital, taken at intervals of ten years on the same patient.

Fig. 65.—Hand of a young girl, aged thirteen years, with congenital symmetrical palmar and plantar keratoderma.

Familial keratoderma is met with either isolated, or associated with generalized ichthyosiform hyperkeratosis, of which it represents merely a localized variety; in its course it resembles symmetrical erythro-keratoderma with which it may coincide.

When essential keratoderma is not strictly congenital, it may develop between the first months of life and the second year, or even in late childhood. The onset is not easily discovered; it is preceded by redness, hyperidrosis and exfoliation in fine lamellæ.

The cases of delayed appearance are distinguished by several authors under the name of *symmetrical keratoderma of adults*, *acrokeratoma*, or *essential tylosis*. The term of Méléda disease comes from an island in the Adriatic, where Neumann and Ehlers found this malformation to be endemic. In the non-hereditary cases, the origin has been referred to consanguinity of the parents.

Palmar hyperkeratosis is serious on account of its incurable character and the resulting inconvenience, but the condition is considerably relieved by moist or rubberized dressings and by keratolytic agents. Radiotherapy, which constitutes the best treatment, has led to noteworthy improvement, but to no complete cure in my experience.

Symptomatic Keratodermas.—Occupational Keratoderma.—This results from friction, pressure or various physical and chemical, chronically repeated irritants, and represents a sort of diffuse callus. The lesions are unilateral or symmetrical and their distribution is often characteristic of certain occupations.

FIG. 66.—Arsenical keratosis. The illustration shows keratotic patches, similar in type to others situated over the forearms, face, scrotum and legs, interspersed with hyperpigmentations and several true epitheliomata. In addition, there occurred a symmetrical keratoderma of the palms and soles. The disease was of twenty-five years' duration and due to drinking water charged with arsenic. (Ormsby.)

Arsenical Keratosis.—Arsenical keratosis, a consequence of chronic arsenic poisoning, is localized especially on the hands and feet. It is often preceded by formication and attacks of desquamative or bullous erythema. It persists indefinitely, even if the arsenic medication is stopped.

It manifests itself under two forms which are sometimes associated: (1) A diffuse yellowish scurfy thickening of the palmar and plantar regions, with well-marked papillary crests; (2) verrucous protuberances which occur in large numbers on both surfaces of

the extremities, sometimes also the face and neck and which may degenerate into arsenical cancer.

Eczema Keratoticum.—The most common keratodermas are those referable to eczema and to syphilis.

Keratotic eczema or corneal eczema of Wilson is usually symmetrical and often of occupational origin. It may be partial or extend over almost the entire region (Fig. 67) and is characterized by its diffuse imperfectly marked borders which pass imperceptibly

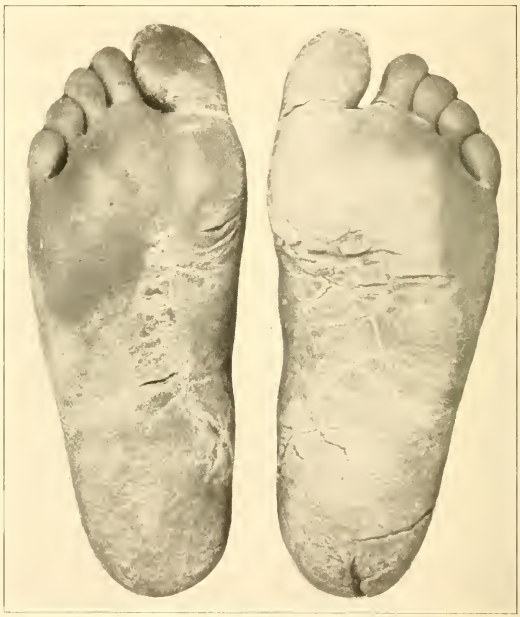


Fig. 67.—Plantar keratotic eczema.

into the healthy skin. It has a tendency to spread along the large grooves. The thickened horny layer splits and becomes exfoliated in lamellæ, exposing a reddened skin, where vesicles are rarely observed. The histological lesions are those of eczema. Foci of eczema or eczematides, are encountered elsewhere, especially on the scalp. The duration of this affection is sometimes several years, with remissions and relapses. It is treated with keratolytic agents

followed by reducing agents; radiotherapy is sometimes very effective.

Palmar and Plantar Psoriasis.—Palmar and plantar psoriasis is usually symmetrical and accompanied by a scattered eruption, but it may be isolated, affecting only a single extremity. It consists at the onset of hyperkeratotic yellowish spots which are distinctly outlined and promptly become exfoliated in dry, friable lamellæ under which the bright red skin is seen. The spots which have

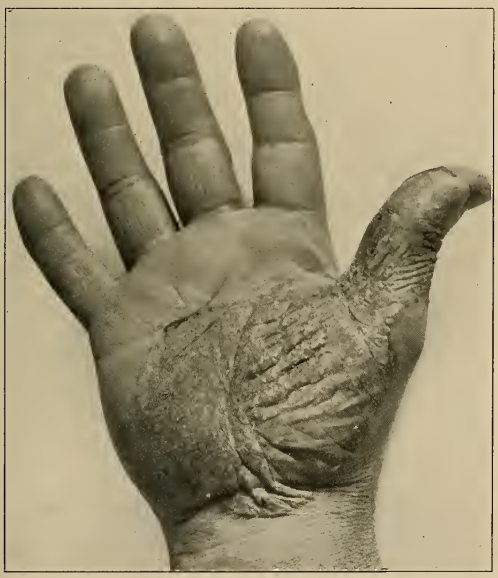


Fig. 68.—Genuine palmar psoriasis.

rounded and clearly outlined contours become confluent in polycyclic patches (Fig. 68). The appearance may be extremely suggestive of syphilis or trichophytosis, but it is rare for these spots not to extend, as manifest patches of psoriasis, to the wrists or to the dorsal regions of the hand, the fingers, or the feet, or for no other spots to appear in different parts of the integument—which clinches the diagnosis. Biopsy shows the characteristic lesions of psoriasis. In this particular localization, psoriasis requires very energetic treatment; sometimes it subsides spontaneously.

In **pityriasis rubra pilaris**, the palms and soles are diffusely reddened, dry, hyperkeratotic and thickened, but desquamation is slight.

Lichen planus gives rise either to small, dry, horny papules, or to depressed more or less confluent keratotic spots (Fig. 23), or to red and finely scaly patches—or finally to total keratoderma.

Gonococcal Keratoderma.—Gonococcal keratoderma manifests itself in the form of horny soles, sometimes a centimeter thick, or as hyperkeratotic or crusted acuminate papules, surrounded by a dark red halo; it affects the plantar and palmar regions, the back of the feet, the lower limbs, sometimes the genitals and exceptionally the upper limbs and the trunk. This affection, which is very uncommon, has been observed especially in connection with gonococcal rheumatism.

Palmar and plantar trichophytosis, elucidated by the work of Djellaleddin Moukhtar, is not, strictly speaking, a keratoderma, but must be mentioned from the diagnostic viewpoint. It appears as perfectly round or polycyclic red spots, surrounded by an epidermic collar. Small vesicles may be discovered on their area and around their circumference. The mycelium is abundantly present in the vesicular fluid and in the scales.

Epidermophytosis of the same regions, more recently discovered, is relatively frequent and presents a variety of aspects. It is therefore essential to keep in mind the imperative necessity of microscopical examination in all more or less doubtful cases.

The reader is reminded that in the course of dysidrosis, or of the pyodermatitides which are sometimes so obstinate in the palmar and plantar regions (*continuous acrodermatitis*), a clinical picture suggestive of various keratodermas is sometimes seen.

The appearance of so-called **punctiform keratosis** (porokeratosis of some writers), characterized by small, sometimes very hard, scattered or grouped, miliary horny masses, may be produced, with slightly variable modifications, by an entire series of skin affections: lichen planus, follicular dyskeratosis, warts, keratotic nevi, familial or arsenical keratoderma, etc.

The **psoriatiform palmar and plantar syphilides**, also erroneously named *syphilitic psoriasis* by some authors, are the most important of all the keratodermas. Bazin grouped nearly all the lesions enumerated above under the heading of palmar arthritides, in order to contrast them with syphilides in the same locality.

These syphilides are relatively common, they may appear at any date between the third month following the infection and the most remote tertiary stages. They resemble each other so closely that it is not always possible to distinguish the premature from the late forms; the latter are described by A. Fournier as "delayed secondary

manifestations." Nevertheless, although certain pictures do not indicate the age of the syphilitic infection, there are others belonging more particularly to one or other of its stages.

In the secondary stage, multiple lesions are especially met with, consisting either of flat lenticular papules of a dusky red color, slightly keratotic and scaly, or of depressed nummular spots, surrounded by a keratotic ridge (Fig. 69); in the tertiary stage and in hereditary syphilis tarda, round or polycyclic spots of a dark or coppery

Fig. 69.—Early secondary palmar syphilide, contemporaneous with the roseola.

red, single or in small numbers, are more usually seen, which may be markedly hyperkeratotic, or wrinkled and cracked, sometimes bordered by tubercles (Fig. 70).

These various lesions are found on any part of the palmar surface of the hand or fingers or the soles of the feet. Though not absolutely diagnostic, a unilateral character of the lesion is somewhat more frequent in tertiary and hereditary syphilis.

The *diagnosis* rests on the sharp limitation of the spots; on their margin of hyperkeratosis with a central depression; on the

infiltration of the base, which is of great value when demonstrable; finally, on the slight tendency of the lesions to encroach upon the neighboring regions. It goes without saying that the antecedents, the co-existence of other symptoms of syphilis, and the Wassermann reaction must be taken into consideration.

Fig. 70.—Late palmar syphilide.

These syphilides are often obstinate and recurrent. *Treatment* with calomel injections, or preferably arsphenamin, often accomplishes their rapid removal, while all other less forcible measures generally fail. Local treatment, with mercurial or with salicylic acid plaster may serve as a useful adjuvant.

Moreover, in all keratodermas, one must never neglect to soften and remove the horny layer (by means of moist dressings, inunction with potash soap, or salicylic acid preparations), in order to open the way for or otherwise assist the special medication that is indicated.

KERATOSIS OF MUCOUS MEMBRANES.

The mucosa of the buccal cavity and the genital organs possess, like the skin, a papillary body and a Malpighian layer, but the

granular layer is absent in the latter and the keratinization of the superficial layers is incomplete.

The lining of the red border of the lips, that of the prepuce and glans, as well as that of a large portion of the vulva, has a structure related to that of the epidermis, so that these surfaces are called semi-mucosæ.

Under pathological conditions, these mucosæ and semi-mucosæ may become the seat of white spots or patches due to the appearance in their epithelium of a great abundance of keratohyalin and eleidin and a genuine keratinization. The term of *keratosis* is justified in these cases.

Leukoplakia.—This affection, which is also known as *leukokeratosis*, white spots of smokers—and erroneously called *buccal psoriasis, tylosis linguæ, buccal ichthyosis*, etc.—is the most common form of keratosis of mucous membranes.

Symptoms.—Leukoplakia is almost invariably located in the mouth, exceptionally in the genital regions. In the mouth, it is usually the tongue, on its anterior half, which is involved to the highest degree. In some cases, the lesions predominate on the lateral portions of the back of the tongue, in others on the margins or the middle; or again, the upper surface of the organ is affected as a whole; the lower surface is less commonly invaded.

More frequent, but as a rule not so marked, is a localization of the disease on the internal aspect of the cheeks, in the form of a symmetrically arranged triangle; representing the commissural white spots of smokers. The posterior region of the cheeks, the gums and the palate, are not often attacked by leukoplakia, which is altogether exceptional in the pharynx and larynx.

On the lips, leukoplakia occupies the posterior surface, the free margin, the external red surface, sometimes the commissures, or all these regions as a whole.

In women, leukoplakia may be observed on the vulva, notably on the inner surface of the labia majora, on the labia minora, the prepuce, the clitoris, the vestibule, sometimes in the vagina and around the anus. In men, leukoplakia of the prepuce and glans is rather rare.

The lesions begin with a smooth condition of the mucosa, which is reddened or of an opaline hue. Once established, they assume two different aspects, according to the severity of the case.

Mild leukoplakia is characterized by obliteration of the papillæ and the grooves of the mucosa, which shows a whitish, grayish, bluish or white color, indicative of a change in the transparency of the epithelium. Exceptionally large pinkish papillæ may be seen shining through this smooth whitish veneer.

The lesions are arranged in spots or patches of extremely variable

dimensions, irregular configuration, sinuous, festooned or ragged margins, sometimes sharply outlined, sometimes gradually fading into the normal state. The entire leukoplasic surface may present a uniform appearance or the center may be more opaque and thickened. The confluence of these spots gives rise to patches, unevenly checkered in dark red, gray or white. The keratotic layer, which is always very adherent, cannot be removed by scraping, without exposing the submucosa; but it is apt to desquamate in small opaline shreds, which on the lips, for instance, the patient can pull off with his teeth.

The *severe* or marked form of leukoplakia is connected by intermediate stages with the mild type. The thickening of the mucosa and of its horny coat may become considerable. Either on an already leukoplasic surface, or on a healthy basis, hard, inelastic, pearly or snow-white patches make their appearance, several centimeters thick with gently sloping or steep borders, with a smooth or roughened surface, closely adherent to the underlying tissues. They may become detached spontaneously, at intervals of months or years, but are promptly reproduced. The entire tongue is sometimes held in a cracked "cardboard" leukoplasic case. The cheeks, the vulva, or the glans penis may also be covered with a coat of this kind.

The keratotic patches are often furrowed by folds or fissures; more rarely, they are studded with acuminated horny protuberances. This verrucous variety is especially noteworthy as being frequently the prelude to epitheliomatous changes.

Another complication of leukoplakia, inconvenient and troublesome rather than actually serious, is an obstinate ulceration which I have named *leukoplasic ulcer* and which will be discussed elsewhere in this book (p. 307).

The condition of the mucosa underlying the leukoplakia is difficult to determine. In the case of the tongue, it is as a rule sclerotic and even retracted proportionately to the hyperkeratosis, either superficially or deeply, as a result of the syphilitic sclerotic glossitis which is the ordinary substratum of the severe leukoplakias. Elsewhere it is often in a more or less marked state of atrophic sclerosis.

The subjective sensations—which are absent in the mild forms of the disease—consist of an impairment of mobility and an unpleasant sensation of dryness and hardness in the severe cases; acute tenderness and shooting pains usually appears only as the result of fissures.

Vulvar Leukoplakia—carefully studied by Jayle and Bender—does not differ from the same disease as seen in the mouth. It may affect the vaginal mucosa and very rarely the uterine cervix. It may precede or accompany kraurosis or sclerotic atrophy of the vulva, with which it has been confused.

The course of leukoplakia is altogether irregular. It usually progresses slowly and persists throughout life. It may remain entirely stationary under the influence of proper hygiene, or it may even subside and disappear, at least partially; but it is very prone to recurrence.

Leukoplakia and Cancer.—The gravity of leukoplakia is due to its complications, which are frequent and very dangerous. Fissures, cracks and erosions are very common in the vicinity of carious teeth or as the result of faulty hygiene; they give rise to acute radiating pains and may lead to lymphangitis or to suppuration.

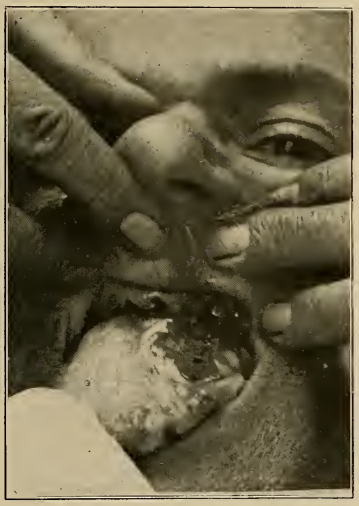


FIG. 71.—Cancer (lobulated epithelioma) on leukoplakia of the tongue.

Epithelioma, however, constitutes the real danger of leukoplakia. It may supervene in all the forms, especially the most severe types, and at any stage of the leukoplakia. Its relative frequency has been variably estimated at 30 and even above 50 per cent. of the cases; a ratio of 15 to 20 per cent. seems to me nearer the truth. [This estimate which seems to me very high applies, of course, only to the severe cases.] The physician may save his patient from an awful death, such as supervenes in cancer of the tongue, if he manages to discover in time the incipient complication and is sufficiently energetic and convincing to obtain consent to a timely operation.

Cancer of the tongue on a basis of leukoplakia (Fig. 71) is almost invariably of the lobulated or spinocellular type and of the variety which I call cancroid; exceptionally, it is of the tubular type. It has two principal modes of onset.

In most cases it begins with a circumscribed, lenticular or more extensive papillomatous elevation, often encircled by a keratotic margin, with a very slightly indurated base; under this superficial form it spreads on the surface during a few weeks, a month or two, before invading the depths of the tissues, so that prompt operative interference is not infrequently successful.

More rarely the epithelioma develops deeply from the start, originating from a fissure which has persisted one or two weeks and at whose level palpation reveals a minute circumscribed woody induration. In these cases, surgical intervention can hardly come in time. Leukoplasic ulcers only exceptionally give rise to cancer.

On the lips, cheeks and genitals, epithelioma appears under entirely similar conditions.

I have emphasized for many years the line of conduct to be followed when cancer is suspected in the course of a leukoplakia; and increasing experience causes me to be more and more positive in this respect.

What should not be done, is: (1) to wait until the symptoms become more pronounced, the epithelioma develops and the glands become enlarged; (2) to resort to an antisyphilitic "therapeutic test" which causes the loss of valuable time; (3) to irritate the suspicious lesion with cauterization of any kind, diathermia, etc.; (4) finally, to try radiotherapy or radium, which are inefficient or even harmful in these cases.

What should be done: in all cases where the diagnosis is not absolutely certain, a specimen removed from the living subject should be at once examined under the microscope, a step which will provide a definite answer in less than twenty-four hours; or, when cancer is known to exist, to proceed at once and without delay to the surgical *extirpation* of the lesion, which affords the only prospect of salvation. [The rich lymphatic tissue of the tongue increases the risk of dissemination as a consequence of the manipulations inseparable from biopsy. After biopsy, therefore, a delay of even twenty-four hours may have serious consequences. In my opinion, the complete operation, whenever possible, should follow immediately on the establishment of the diagnosis which should therefore be made on frozen sections, with due consideration of the difficulties of this method.]

Pathological Anatomy.—On a leukoplasic mucosa (Fig. 72) the rete Malpighii is greatly thickened (acanthosis) and its interpapillary plugs are hypertrophied in all directions; between the mucous body and the extremely thick horny layer which covers it, and which

consists of cells deprived of their nuclei, a granular layer has made its appearance, with very abundant keratohyaline and eleidin, diffusing into the stratum corneum; these lesions explain the whiteness of the patches.

In the papillary body of the corium is seen a variable infiltration of round cells around the vessels, as well as sometimes the lesions of endo-perivasculitis and connective-tissue sclerosis.

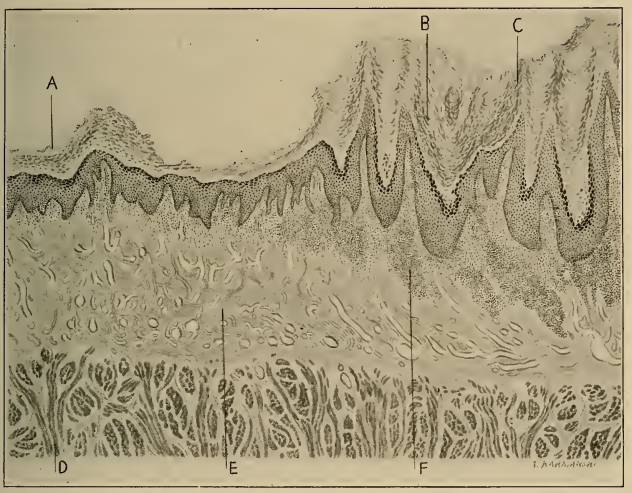


FIG. 72.—Simple and verrucous leukoplakia of the tongue. Under the epithelium, which is greatly thickened and abundantly provided with keratohyalin and diffuse eleidin, is seen an inflammatory cellular infiltrate, especially abundant at the level of the verrucous portion; a newly formed layer of sclerotic tissue, traversed by numerous dilated bloodvessels, has become interposed between the epithelium and the muscular tissue. No trace of epitheliomatous change is demonstrable. *A*, simple leukoplakia; *B*, eleidin; *C*, verrucous leukoplakia; *D*, muscular tissue; *E*, sclerosis; *F*, inflammation. × 18.

When epitheliomatous transformation occurs, it results from atypical budding of the interpapillary processes or of the epithelium which lines the fissures (Fig. 192). Invasion of the lymph spaces takes place very rapidly, especially in the tongue.

Etiology.—Leukoplakia is at least ten times more frequent in men than in women. It is observed especially from thirty to fifty years of age, but cases have been reported at the age of twelve years (Bénard) or even younger. I have encountered it, complicated by cancer of the tongue, in a girl of nineteen years, which is excep-

tional from every point of view. Leukoplakia may result from multiple causes of different kinds. The equation:

Leukoplakia = syphilis + tobacco

although often true, is certainly too absolute. Tobacco is the most powerful of the local causes; but dental lesions, false teeth, abuse of alcohol and condiments, etc., also play a part in the production of buccal leukoplakia, especially in non-smokers and in women. Other irritants intervene in genital and anal leukoplakia.

In the great majority of the cases, leukoplakia develops on a syphilitic soil, but I believe that, even on the plea of hereditary syphilis, it is not justified to consider it as an invariably parasyphilitic affection, as maintained by Professors Landouzy and Gaucher. There are cases where syphilis is absent, where the Wassermann reaction is negative and where nothing can be suspected except local irritative factors, obscure auto-intoxications, or perhaps an individual predisposition.

At the present state of our knowledge it is impossible to say if it is correct to recognize a syphilitic leukoplakia, and other nicotine, dental, post-infectious leukoplakias, differing by their symptoms and their course, or if leukoplakia is a syndrome resulting from various causes, a modification of the epithelial evolution resulting from infectious or toxic vascular lesions. The last-named theory is the most probable in my opinion.

Treatment.—This must be carefully handled and requires in the first place a strict buccal *hygiene:* absolute abstinence especially from tobacco, from alcohol, condiments and highly spiced foods, irritative mouth-washes; the teeth must be put and kept in perfect condition.

Soothing or weakly alkaline irrigations of the mouth with Vichy water, or Saint Christau water, are useful palliatives, preferable to pastils and compressed tablets. The warm mineral springs of Saint Christau, with local spraying, or to a less degree, various sulphur springs, yield favorable results.

Among *topical* applications, those most to be recommended are glycerol salves or ointments made with balsam of Peru, oil of cade, oil of birch, or salicylic acid. In case of thick keratotic patches, their detachment may be furthered by painting with acid nitrate of mercury, chromic acid, or potassium bichromate, etc. Sometimes, the destruction of certain patches with the thermocautery, or their surgical removal, may be called for. In a general way, however, *caustics are not to be trusted,* especially in case of fissures, which patients like to treat and abuse with silver nitrate; cauterizations are certainly more injurious than useful. Radiotherapy and radium,

energetically employed, have a few cures to their credit, but on the other hand a large number of failures are on record.

The operative indications in case of probable or certain cancer need not be repeated here.

General treatment with antisyphilitic agents, is evidently required in the presence of specific antecedents or a positive Wassermann reaction; but it has been found effective even in cases where syphilis was apparently excluded. Intragluteal calomel injections exert such a preponderating action in leukoplakia that they were accorded the preference until the introduction of arsenobenzol; the latter is still more active, especially in combination with soluble mercurial injections. In obstinate cases, I have often been excellently served by local treatment with injections of very dilute cyanide of mercury. Iodides are not to be recommended. When an established leukoplakia in a syphilitic patient has only partially yielded to specific treatment, or resisted it, the advantage will at least have been served of opposing the extension of the disease and preventing the appearance of other syphilitic manifestations.

General hygienic recommendations must not be neglected, especially in regard to diet, correcting dyspepsia if necessary, or controlling any auto-intoxications which may have been discovered.

There is a certain class of leukoplasic patients whose life is made miserable by a regular "cancerophobia," liable to be maintained by treatment of any kind. In these cases, a judicious psychotherapy is indicated.

Syphilitic Glossitis.—It seems to me useful to describe in connection with leukoplakia the lesions which secondary and tertiary syphilis so frequently produce upon the tongue. This organ may present:

1. The *smooth patches* of Fournier; "mowed-lawn patches" of Cornil. These are pinkish spots, devoid of papillæ, dry, non-indurated, round or oval, distinctly outlined, though without special border. These lesions may be premature, developing rather rapidly, or delayed, and in this case are much more obstinate.

2. *Opaline mucous patches* seen only on the margins or at the tip of the tongue, generally near carious teeth; sometimes they are eroded or fissured; rarely, on the back of the tongue, they are papular or lozenge-shaped.

3. *Hypertrophic* or *papillomatous papules,* with a gray or reddish surface, very rare, occupying the vicinity of the circumvallate papillæ where they become confluent in a large patch; from here they reach the back of the tongue, which then assumes the appearance called "toads' back."

4. *Tuberculo-ulcerative syphilides* and *gummas,* which will be discussed elsewhere; these may leave sclerotic cicatrices.

5. Finally, *sclerotic glossitis*, of the tertiary period, very common, especially in men; it may be superficial or deep.

In the *superficial* form there are a few *islands* of cortical sclerosis, or a *single* smooth red surface, with lamellar induration, sometimes complicated by fissures.

Deep glossitis, occupying especially the middle or the borders of the tongue, or its entire anterior half, is characterized by an irregular lobulation, prominent papillæ being separated by deep grooves united in a network; fibrous induration of the organ is also demonstrable (Fig. 73). The mucosa is wine-red in some places, discolored in others, smooth, tense, practically devoid of papillæ throughout.

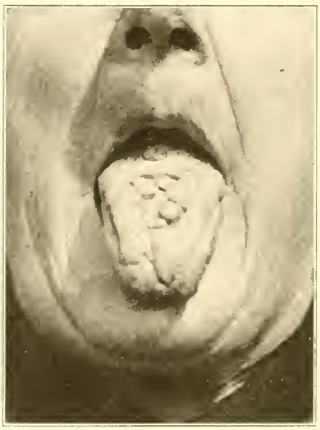


Fig. 73.—Sclerotic syphilitic glossitis of the deep variety, in a woman aged sixty years.

The coexistence of syphilitic lingual sclerosis and leukoplakia is very common.

Sulcated or Scrotal Tongue.—Sclerotic glossitis must not be confused with a congenital, often familial, malformation which bears this name. The organ in such cases is lobulated and fissured; its very villous surface is studded with prominent fungiform papillæ. But its consistence is soft, there is no pain and the condition is absolutely permanent.

Median Rhomboidal Glossitis.—This affection was described in January of 1914 by Brocq and Pautrier; it is not rare and I have seen numerous instances. The dorsal aspect of the tongue, in front

of the circumvallate papillæ presents a reddish raw surface; the lesion is usually mammillated, slightly indurated, indolent, and extremely persistent. Anatomically, nothing is found but hyperacanthosis and a moderate infiltration of the corium. This glossitis, which occurs especially in adults, is often wrongfully ascribed to syphilis; its nature is unknown; it resists all local or general treatment.

Lichen Planus of Mucous Membranes.—This is very commonly confused with leukoplakia or with syphilides. Its diagnosis nevertheless is relatively easy.

Lichen planus of the mucosæ, especially of the mouth, is not rare. It is observed in about one-half of all lichen cases. It may persist beyond the cure of the cutaneous manifestations, or it may constitute a primary and sometimes exclusive localization. Familiarity with its characteristics is therefore necessary.

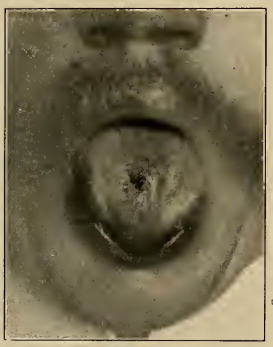


FIG. 74.—Lichen planus of the tongue in a man aged forty-two years.

The seat of election of lichen planus of the mucosæ is in the first place on the internal aspect of the *cheeks*, at the level of the alveolar margin, preferably opposite the last molars. It manifests itself on one side, or symmetrically, in form of one or several *white patches* with an irregular reticular or annular arrangement resembling lacework, pure white or bluish in color, on a normal background; the more or less delicate network is crossed by larger bars. Erosion or desquamation never occurs.

Next in order is the localization on the *tongue* (Fig. 74), which may present either patches of a dull bluish white, smaller than a lentil, with a reticular or fern-leaf design, or opaline surfaces through which a few rose colored papillæ project.

Lichen planus may furthermore be met with on the lips, palate, gums, tonsils, on the glans and prepuce, where it is often annular, etc.

These lesions are absolutely sluggish and indolent; they persist for months, or oftener for many years, undergoing very gradual changes.

Histology shows a thickening of the epidermis in all its layers, the appearance of a stratum granulosum, dome-shaped papillæ, and a cellular infiltration in the papillary body. The structure is accordingly entirely that of lichen planus of the skin. The explanation of the white color is the same as for the network of the cutaneous papules of typical lichen planus; it moreover assumes an identical form in the majority of cases.

The *treatment* of lichen of the mucosæ need not be very active, the condition being obstinate but by no means serious. It never leads to cancer. Soothing mouth washes, inunction with balsam of Peru, painting with potassium permanganate, 1 to 100, are quite sufficient. High frequency currents and radium therapy have not yielded favorable results in my experience. A bland diet, good hygiene, a quiet life, arsenic internally, are recommended.

Lupus Erythematodes.—It is exceptional for lupus erythematodes to affect the mucous membranes. It is sometimes seen to radiate in the form of a scaly redness on the posterior aspect of the lower lip starting from the free border. When it develops on the internal aspect of the cheeks or on the tongue, it manifests itself in the form of a circumscribed patch, mottled with white and bright red, and partly atrophic.

Smooth Patches of the Tongue.—Occasionally, the tongue is seen to present spots or large patches devoid of papillæ, reddish and glistening, without a white margin, absolutely stationary during months or years. They are entirely indolent and the patient may be unaware of their existence. In other cases, they are very sensitive, and associated with *glossodynia*.

These patches are always suggestive of the cropped patches of syphilis, or of incipient leukoplakia. I am personally convinced that they may be referable to a variety of causes, notably tuberculosis, diabetes, chronic dyspepsias, severe neuropathies; sometimes they are connected with carious teeth or badly fitting artificial dentures.

Those cases in which desquamation and a varnished appearance are extensively combined, or involve almost the entire tongue, are known under the name of *red tongue*, or *glossitis* of *nervous arthritics*.

Glossitis Exfoliativa Marginata.—This peculiar affection which also bears the names of *exfoliatio areata*, or *geographical tongue*, gives rise to circinate patches, rings and other designs upon the tongue. The spots are composed of a white border, one or two millimeters wide, within which lies a desquamated surface, bright

red near the margin, gradually fading away at a distance from it. More or less rounded at first, the spots spread rapidly and become confluent with their neighbors; the resulting design may thus become entirely altered in a day or two. New spots are incessantly reproduced. There is no induration of the mucosa.

Entirely indolent, usually ignored by the bearer, this affection lasts for years or indefinitely. Its etiology is unknown. It is observed in children and in adults. It is often familial. Sulcated tongues are specially predisposed to it. In spite of the parasitical appearance of the lesions, no case of contagion has been reported. Confusion must be guarded against, especially with syphilitic lesions. Parrot referred exfoliative glossitis to congenital syphilis, which is certainly incorrect.

[**Chronic superficial excoriation of the tongue,** or *Mœller's glossitis,* is characterized by irregular circumscribed intensely red spots of great chronicity, without tendency to extension or ulceration, located in the middle or the anterior portion of the tongue. Contact with food usually provokes severe burning or lancinating pain.]

Black (Villous) Tongue.—This is a lingual keratosis, in which the filiform papillæ, far from becoming obliterated, as in leukoplakia, became immediately elongated and assume a dark, black or brown color.

The affection begins on the middle line, not far from the circumvallate papillæ and spreads forward and to the sides, remaining more pronounced at its point of origin; the borders of the lesion are diffuse. The papillæ, which may reach the length of a centimeter, lie like mown wheat and a parting may be made as in the hair (*black hairy tongue*). The brown color is darker at the extremity of the papillæ.

When these papillæ are excised, the microscope shows an enormous hypertrophy of their corneal sheath, the lamellæ of which adhere to the axis and spread away from it like the branches of an old pine tree. The presence of eleidin has been reported at the border of the rete. The black color is due, not to foreign bodies or to pigment, but to a smoky tinge of the horny substance itself, as in black ichthyosis.

This rather uncommon affection is observed in adults and aged individuals; I have seen it associated with *pharyngo-mycosis.* It proceeds in attacks, followed by partial desquamations; it is sometimes prolonged for months or years and has a tendency to recurrence.

Black tongue is neither contagious nor inoculable. It has been suspected of being due to a special infection by a microbe or a yeast. But the results of investigations which have been undertaken to prove this are entirely contradictory and the parasitical theory can no longer be maintained (Lebar, 1917).

There are cases of villous tongue, presenting identical features, but without a brown or black coloration.

This affection must not be confused with a simple coated tongue, with an accidental discoloration due to articles of food or medicinal agents, with the pigmentation of Addison's disease or that of argyria. Mouth washes with peroxide water sometimes produce at the end of a certain time an appearance identical with that of villous black tongue; but the surface promptly returns to the normal when these washes are omitted. In several great dystrophies, such as acanthosis nigricans and psorospermosis, the tongue is villous in its entire extent, but of a normal or whitish hue.

The *treatment* consists of alkaline mouth washes and painting with a 5 per cent. or 10 per cent. alcoholic solution of salicylic acid.

DYSKERATOSES.

The dermatoses which I group under this heading are characterized by a faulty development of the epidermis, in the course of which a certain number of Malpighian cells become differentiated from their fellows, undergoing an abnormal, premature and imperfect keratinization. These dyskeratotic cells are present in the layers of the epidermis, as far as in the horny layer, in the form of

FIG. 75.—Round bodies and granules of follicular psorospermosis. *a* and *b* round bodies, Malpighian cells surrounded by a membrane and containing granulations of keratohyalin and a nucleus; *c* and *d*, granules, nucleated dyskeratotic cells which have completed their development.

"round bodies," "granules" (Fig. 75), globes or corpuscles, with or without nuclei, easily distinguished from the still normal cells as well as from the parakeratotic cells.

The group of dyskeratoses comprises: (1) The disease which I had named *psorospermosis follicularis vegetans;* (2) *Paget's disease of the nipple*; (3) the *precancerous dermatosis* of *Bowen*, or *lenticular and discoidal dyskeratosis.*

From the strict viewpoint of the histological structure, *molluscum contagiosum* should likewise be classified with the dyskeratoses;

but this affection is altogether different from the preceding and will be discussed with the benign tumors.

Dyskeratosis and analogous cellular changes are also observed in several forms of cancer, notably in the majority of spinocellular epitheliomas, where they were formerly described under the name of physalides (Virchow) and more recently under that of *pseudo-coccidias;* and two dyskeratoses, Paget's disease and Bowen's, take first rank among the precancerous affections. This fact is interesting and suggestive, but its significance is not clear as long as the cause and nature of dyskeratosis remain unknown.

Darier's Disease.—This important dermatosis which I described in 1889 under the name of *psorospermosis follicularis vegetans,* is generally called *Darier's disease,* especially abroad; the most appropriate denomination for it, in my opinion, would be *dyskeratosis follicularis.*[1]

It manifests itself clinically as papulo-crusts, often follicular, capable of becoming confluent in verrucous surfaces with crumbly margins, located symmetrically in certain definite regions. It is essentially chronic and almost incurable.

The improper name of psorospermosis which I had attributed to it, was based on an erroneous interpretation of the corpuscles which are found in the epidermis and which at the time were mistaken for coccidia or psorospermia, namely for parasites of the sporozoa group. They have now been shown to be really dyskeratotic epidermic cells.

The *etiology* of this dermatosis is unknown, it has been observed in all countries, but is relatively rare; it seems to affect the male sex somewhat more frequently; its familial and hereditary character is apparent from numerous observations, but there is nothing to indicate contagiousness. It has been compared with ichthyosis, the icthyosiform hyperkeratoses and acanthosis nigricans; there is a tendency to regard it as a dystrophy of the epidermis of congenital origin.

The typical *eruptive lesion* is a papule covered with a grayish brown crust, having the dimensions of a pin-head to a small lentil. On removal of the hard and horny, prominent or flattened, rather adherent crust, it is found to be imbedded in a funnel-shaped depression with raised margins, into which it sends a soft yellowish process, of sebaceous appearance; this depression is the dilated orifice of a pilo-sebaceous follicle. There are other lesions, however, which are not follicular.

At the onset the patients notice a dirty tinge and roughened state of the skin; later on, the crusts become confluent in warty patches.

[1] In the original this section is headed Psorospermosis, etc. The editor has substituted the name by which the disease is generally known.—S. P.

In the groins, the axillæ and in all moist regions, pinkish globular or crater-shaped *vegetations* may develop, in exceptional cases, arranged in cauliflower or fungoid masses, having an offensive odor.

The *eruption* is symmetrical and occupies large areas. Its sites of election are: the face, especially the temples and the naso-labial

Fig. 76.—Follicular dyskeratosis [Darier's disease]; general distribution of the dermatosis.

fold; the scalp, which becomes crusted and moth-eaten, but without alopecia; the conchæ of the ears, the presternal and interscapular grooves; the belt line, the perigenital region and the large articular folds. This topographical distribution is in the main that of seborrhea (Fig. 76); the entire trunk and the external aspect of the

limbs may be likewise involved. So-called abortive or incomplete cases are not very uncommon.

On the back of the hands, elevations identical with flat warts are often seen; the palmar and plantar regions present a punctate keratosis composed of yellowish translucid points; the nails are striated and brittle; the tongue may be villous.

The disease begins, in one-half of the cases, between eight and sixteen years of age (Fig. 77), but also earlier or later; sometimes at the temples and face, sometimes in the groins; it spreads rapidly and then remains indefinitely stationary. It is accompanied by no subjective symptoms and interferes in no way with the general health.

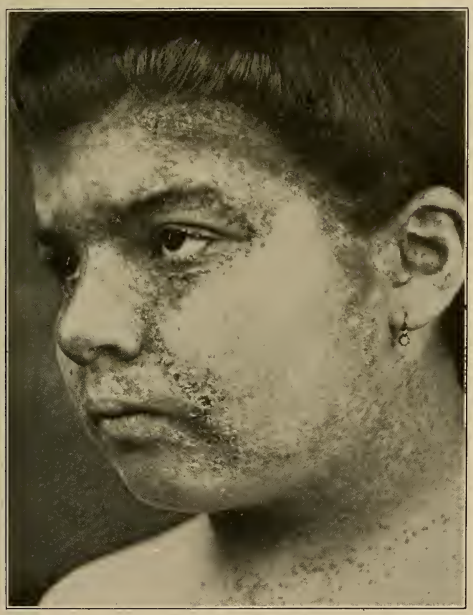


Fig. 77.—Follicular dyskeratosis [Darier's disease] in a girl aged eleven years.

The *pathological anatomy* is characteristic. The illustration (Fig. 78) conveys a sufficient idea of the condition.

The microscopical demonstration of the granules in the crusts which are easily removed from the patient, and of the round bodies in the underlying substance, is very easy and suffices for a positive confirmation of the clinical diagnosis.

Without curing the psorospermosis entirely, it is possible to improve the patient's condition very markedly by baths and soap washes, inunctions with salves containing keratolytics, followed by reducing agents as well as by means of radiotherapy.

Paget's Disease of the Nipple.—This is a chronic affection which develops on the nipple and areola of women past forty years of age; or, exceptionally in men in the perineo-scrotal and some other regions. It seemes to be less uncommon in England and in the United States than in France.

FIG. 78.—Histology of follicular dyskeratosis; section through a perifollicular papulo-crust. *a*, orifice of a hair-follicle; *b*, *b*, hairs, and *c*, sebaceous gland, unchanged; *d*, thickened horny layer containing (in *e e*) collections of granules; *f*, *f*, granular layer interrupted at the level of the foci of dyskeratosis; *g*, *g*, fissures and lacunæ, resulting from fibrino-mucous disintegration of the epidermis, in which a few granules are seen floating (*h*, *h*); *i*, *i*, round bodies.

At the onset, only a few small crusts are seen on the nipple, with warty elevations and sometimes a serous oozing. After some months or years, a progressively extensive erosion makes its appearance and may invade the entire integument of the breast. At its fully developed stage, there exists an eroded or ulcerated red patch, mottled with pinkish epidermis-covered islands, remarkable for its distinctly outlined polycyclic contours, which are bordered by a narrow margin or cuff of scales (Fig. 79). The eroded surface

presents a very distinct parchment-like induration; it never undergoes retrogression or spontaneous cure. The nipple is retracted. The lymph glands are not usually enlarged. Finally, sometimes after a considerable number of years, a malignant neoplasm may supervene in the form of a hard superficial or deep node which ulcerates and presents the usual appearance of cancer of the lactiferous ducts, or of ordinary cancer of the breast.

The *differential diagnosis* from eczema of the mammary areola, which is practically limited to the puerperium or to cases of scabies (Fig. 157), has less distinctly circumscribed borders and takes an acute course, is easy. Certain superficial epitheliomas, which simulate Paget's disease, can be differentiated only by means of biopsy.

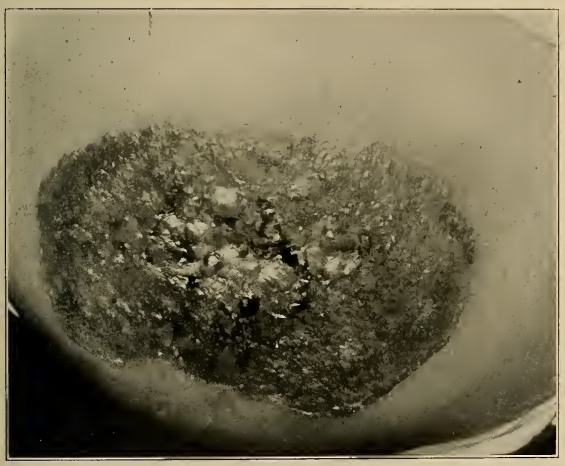


Fig. 79.—Paget's disease of the nipple. After a cast in the Museum of the St. Louis Hospital, Paris.

The *histology* of Paget's disease shows a Malpighian layer interspersed or even packed with vacuolated cells and round bodies, as well as cells with deformed, sometimes monstrous nuclei; their large numbers as well as the acantholysis of the intermediate cells, gives the epidermis a disorganized appearance and leads to the production of oozing or crusted erosions. The scales which persist, appear under the microscope dotted with vacuoles and pseudococcidia. The papillary body is infiltrated with plasmocytes, sometimes arranged in a continuous layer.

The threatened contingency of cancer necessitates the *treatment*

of Paget's disease by [radical] surgical measures. Radiotherapy has been tried, without success.

Precancerous Dermatosis of Bowen or Dyskeratosis Lenticularis et Discoides.—I have recommended that the name of J. T. Bowen be attached to an essentially chronic and progressive dermatosis first described by him in 1912. It appears in the form of lenticular or nummular disks (Fig. 80), later patches of irregular shape, covered with thick scaly crusts; the lesions are generally multiple, non-symmetrical, and may be situated anywhere. Atrophic spots, resembling Paget's disease, may also be seen.

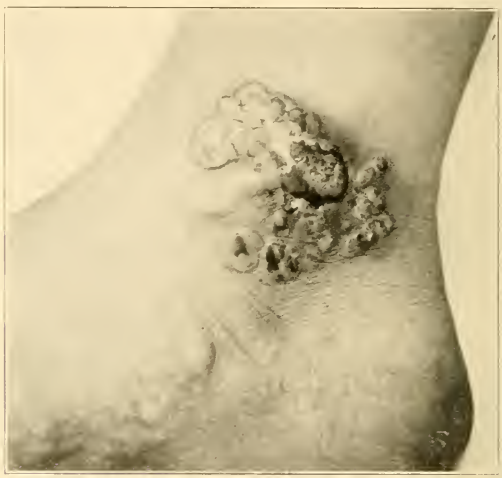


FIG. 80.—Dyskeratosis of Bowen, showing a patch composed of lenticular and discoid lesions on the internal malleolus of a woman, aged thirty-nine years. Personal observation, 1914.

The histological structure of the lesion is entirely analogous with that of Paget's disease, especially in regard to the dyskeratosis and the vacuolization; but in Bowen's disease a well-marked hyperkeratosis is demonstrable. The differential diagnosis from psoriatiform eczematides, tertiary syphilides, psoriasis, senile keratosis lupus erythematodes, certain forms of lichen, etc., must be confirmed by biopsy.

In one-half of the reported cases, the affection was observed to degenerate into cancer. Hence, there is good reason for expressly recommending the total destruction, or better, the surgical removal of the diseased areas.

CHAPTER XII.

PAPILLOMATOUS AND PROLIFERATING DERMATOSES.[1]

Papillary proliferations are more or less prominent excrescences, which may be conical, filiform, or resembling a cauliflower growth, arranged in patches or spread out over large areas.

They must not be interpreted as resulting simply from the *elongation* of preëxisting normal *papillæ;* on a given surface the normal papillæ are actually infinitely more numerous than the excrescences which can lodge there. Each proliferation really corresponds to several papillæ, united on the same axis or connective tissue vascular stalk.

The anatomical conditions which give rise to the phenomenon of papillary proliferation can be explained in three different ways: It may result from an active primary proliferation of the upper layer of the derma known as the papillary body, or from a primary proliferation of the rete Malpighii; or from a simultaneous hypertrophy of these two layers. As a general rule, as was demonstrated by Auspitz, the Malpighian hyperplasia, known as acanthosis or hyperacanthosis, is the original disturbance. It is not understood why this gives rise sometimes to a simple epidermic papule (for instance, the flat juvenile wart), sometimes to a papillomatous elevation (for instance, the papillary wart).

The horny layer which covers the proliferations may have its normal thickness, or it may be thinned, as in the venereal warts; or on the contrary, thickened as in common warts. Hence, there are *naked proliferations* with a smooth pink surface; others have the normal color of the skin; still others are of a grayish yellow color, firm consistence, and evidently hyperkeratotic. The latter are named *verrucosities* or *verrucous excrescences.*

No very distinct boundary line can accordingly be established between the two dermatological forms of keratoses and verrucosities.

As regards the term *papilloma,* this approximately corresponds to

[1] In the original, this chapter is headed végétations et dermatoses végétantes. These terms cannot be literally transcribed into English. The term végétation connotes a papillary proliferation for which the word papilloma was formerly used. But as Virchow long ago pointed out, this term is unscientific; the condition is not a tumor of the papillary body but rather a proliferation of the interpapillary epithelial processes with secondary changes in the papillæ. The adjective papillomatous, however, may properly be used to indicate this clinical condition.—Ed.

that of vegetation, and like the latter does not designate a dermatological type or species, but an objective appearance which may be brought about in a variety of dermatoses and tumors.

Papillomatous Dermatoses.—These can be divided into three groups:

A. **Essential Papillomatous Dermatoses.**—Some are circumscribed, like the venereal warts, which will be discussed in a special paragraph; the same is true for *common warts.*

The verrucous nevi have been mentioned in connection with the circumscribed keratoses and will figure again among the nevi in general (XXXI).

Others are *generalized* or *regionally diffused.* It is sufficient to recall the hystrix varieties of ichthyosis and generalized hyperkeratosis. Under this heading belongs a peculiar cutaneous dystrophy, known as acanthosis nigricans.

B. **Accidentally Papillomatous Dermatoses.**—Several skin diseases, of infectious, toxic, or indefinite character, may present proliferations as a form of eruption, as an accidental manifestation, or as a developmental phase. From the diagnostic point of view, it is important to compare them with each other.

C. **Tropical Proliferating Dermatoses.**—The establishment of this group may appear unscientific; but it is justified in the present state of our knowledge and, moreover, is convenient.

VENEREAL WARTS.

Also named *condyloma acuminatum* and popularly known [in France] as cauliflower growths, cock's comb, etc., venereal warts are agminated papilliform excrescences, of a pinkish or grayish color, having their seat of predilection about the genital organs and the neighboring folds.

In *men,* they are almost exclusively situated in the glando-preputial groove, on the corona glandis and the frenum, but may affect the entire prepuce and the urethral orifice.

In *women* (Fig. 81) they affect the vestibule of the vulva, the fourchette, the preputium clitoridis and sometimes cover the entire vulva, the genito-crural folds, the anus and the intergluteal region.

At the onset, the lesions are simple pinkish granules or branching elevations, like a mole's paw; as they grow they form tufts of filiform or lamellar processes, sometimes reaching a length of several centimeters. Whether sessile or pedunculated, the warts originate on a healthy unthickened skin or mucosa, which is sometimes irritated or macerated. In women who are neglectful of cleanliness, especially in case of gonorrhea and pregnancy, they finally form enormous mammillated masses the size of a fist, bright red in color,

oozing and offensive. They cause inconvenience but no actual pain. They may be found very rarely in the axillæ and on the scalp, but not elsewhere.

The *histology* of these growths shows a very pronounced hyperacanthosis, with abundant karyokineses, covering filiform and branched connective-tissue protuberances which are traversed by bloodvessels with large lumina. There may be no suggestion of acute inflammation in these tissues. The granular layer is discontinuous, the horny layer is very thin.

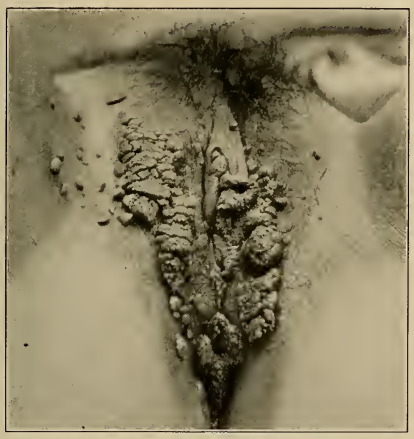


Fig. 81.—Venereal warts of the vulva.

The *etiology* has not been elucidated; these growths are reputed to be contagious and auto-inoculable; on the other hand, I have certainly known them to appear without direct contagion, under the influence of ordinary irritations, gonorrhea, pregnancy, etc. Their kinship to common warts has been frequently suspected, but has not been demonstrated.

Treatment by attention to hygiene, astringent lotions, bland powders, pulv. Sabinæ with an addition of salicylic acid, 2 per cent. will usually cause them to wither, though not to disappear entirely. Among caustic agents, pure carbolic acid, or chromic acid, may prove successful when the warts are very small.

Voluminous growths require ablation with the scissors, or detachment with the forceps, or better with a curette; the operation is rather painful. Anesthesia, local or general, is sometimes necessary;

inferior spinal analgesia with cocain, which deprives the region of all sensation, would be very valuable in extreme cases of profuse and exuberant growth. My assistant and friend Dr. Chicotot has successfully removed large masses of condylomata by means of radiotherapy.

VERRUCA VULGARIS.

A common wart is a papillary hyperkeratotic excrescence; it is typical of products of this sort, which are accordingly known as warty or verrucous.

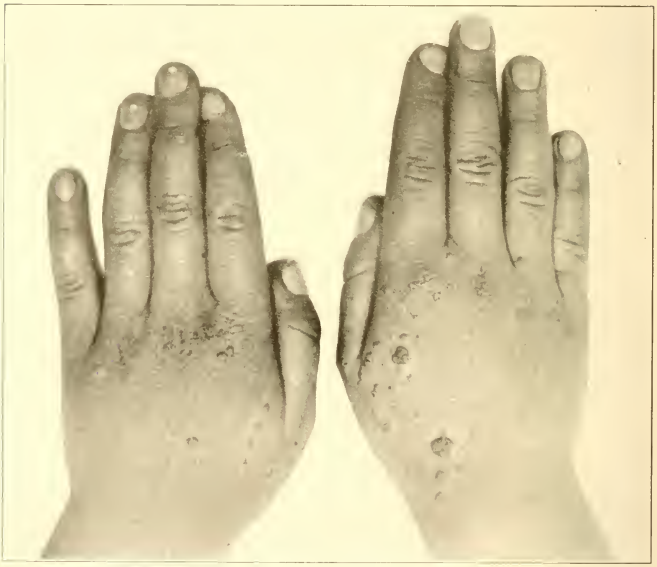


FIG. 82.—Common warts on the hands of a school-boy. Note near the left thumb a trail of four warts which developed on a scratch made with a pin.

A wart consists of a rather prominent, rounded and distinctly circumscribed elevation, from the size of a pin-head to that of a small bean, usually as large as a pea, of a grayish, yellowish or gray black color, with a mammillated surface sometimes studded with villous protuberances, of a hard rough consistence. The surrounding skin is not congested. Some warts are constricted at their base, almost pedunculated, while others spread out and are not much raised. They are not sensitive or painful except at the borders of

the finger-nails and in regions exposed to pressure, notably on the soles of the feet.

Warts are usually multiple and have their seat of election on the dorsal or lateral aspects of the fingers and the hand (Fig. 82), where they may become confluent in patches, they also occupy the periungual or subungual groove; more rarely; the palm of the hand or fingers, the face, the eye-lids, the scalp and the soles.

Juvenile flat warts are probably only a special form of the same affection (p. 130).

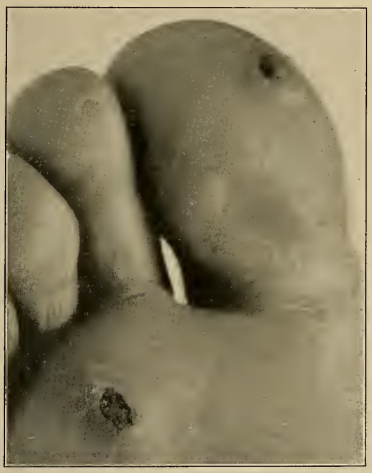


Fig. 83.—Plantar warts in a girl aged twenty years.

Plantar warts, described by Dubreuilh and M. Robert, are noteworthy on account of their extreme tenderness and the special treatment which they require. They are preferably situated on the supporting points of the foot and at first sight resemble calluses, but on closer examination, they are seen to be usually composed of filiform growths, arranged in a cluster and surrounded by a raised border (Fig. 83). They can be cured without much difficulty by means of radiotherapy. [I have encountered some plantar warts which are extremely resistant to treatment and have yielded only to the actual cautery or to applications of carbonic acid snow after the removal of the thick horny layer by means of strong salicylic acid plasters and curettage.]

All warts may become fissured and inflamed.

These formations are extremely common and are observed especially in schoolboys, in youthful individuals doing manual labor, but also in adults. The contagiousness and auto-inoculability of warts, always asserted by the laity, have been proved beyond a doubt by the experiments of Variot and others; the incubation lasts longer than a fortnight. The microbe is unknown; the *bacillus porri* which has been described, has not been proved to be the causative agent. [Wile and Kingery have reproduced epithelial hyperplasia by injection of the filtrate made from the substance of common warts.]

The *histology* of warts shows an enormous increase in the length of single papillæ or of groups of papillæ and of their vessels and a considerable thickening of all the layers of the epidermis; there is no inflammatory infiltration in the cutis.

The *treatment* must aim at the avoidance of cicatrices, for the warts often disappear spontaneously. It is most advisable to cauterize them carefully with the galvanocautery, or with a very fine point of thermocautery, assisted by the curette. Carbonic acid snow, properly applied, is likewise very successful. Radiotherapy gives remarkable results, without pain; but it requires great caution, especially on the back of the hand.

Electrolysis has been recommended in case of large warts and magnesium ionization when they are very numerous; and, very recently, solar light condensed by means of a large lens. It is difficult to determine the efficacy of the daily administration of 75 centigrams of magnesium, or of a few drops of tincture of thuja, or of arsenical medication.

Nitric acid does not deserve the favor which it enjoys, for it has caused innumerable burns and distressing scars. Less powerful caustic agents, collodium, plasters, etc., are apt to be disappointing. The juice of the milk-wort and of the celandine, very persistently applied, causes the warts to wither and drop off.

These various measures may prove successful, but it is important to keep in mind a surprising but undeniable fact, stated by many writers, namely, that simple suggestion may have the same effect.

[After persisting for a variable period, months or years, warts generally disappear without any known reason.]

PAPILLARY AND PIGMENTARY DYSTROPHY OR ACANTHOSIS NIGRICANS.

This disease was described under the latter name by Pollitzer and Janovski, in 1890, after I had observed and named it papillary and pigmentary dystrophy [but had not published the observation]. It is characterized by two fundamental phenomena: (1) a roughened condition of the skin, with scattered or agminated papillomatous

elevations; and (2) a dark pigmentation. The lesions are essentially regional, ordinarily symmetrical, with somewhat diffuse borders. In order of frequency, they affect the axillæ, the neck and nape of the neck, the ano-genital region, the internal aspect of the thighs, the fold of the elbow, the popliteal space, the umbilicus, the back of the hands, the areola of the breasts and the feet.

The roughened condition of the integument is due to an exaggeration of the folds and fissures and does not disappear on stretching the skin; from this surface, which is studded with papillary protuberances, of a brown or even black color comparable to the bark of a tree, but supple and sometimes scaly, arise sessile or peduncu-

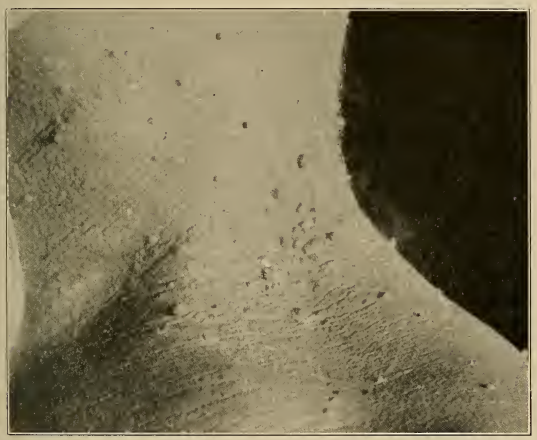


Fig. 84.—Acanthosis nigricans, of five months' standing; portion of the supraclavicular fossa of a man aged forty-two years, suffering from cancer of the liver. After a cast by Baretta.

lated papillomatous proliferations (Fig. 84), isolated or in patches; on the free border of the eyelids and lips, they may present a regular arrangement like the teeth of a comb.

In the palmar and plantar regions, the normal papillary ridges are notably exaggerated. The tongue is nearly always villous, but the mucosæ are never pigmented.

The finger nails are brittle, generalized alopecia is common; pruritus has occasionally been noted.

Acanthosis nigricans is a rare disease, about sixty [at least eighty to a hundred] observations have been reported from all countries, with a slight predominance of the female sex. Its principal interest is

due to the fact that very frequently, in about two-thirds of the cases, it is associated, as was first pointed out by me, with a cancer of the abdominal cavity, primary carcinoma of the stomach or intestines, for instance, or it may be secondary to a cancer of the uterus, or the breast, etc. This dermatosis may accordingly put the physician on the track of a latent cancer, as has happened in several instances.

It begins with a dirty-looking pigmentation of the neck and axillæ, or the appearance of one or several warty growths. The course is rapidly progressive, sometimes interrupted by remissions, and cachexia supervenes in less than a year, two years at most.

This so-called *grave form*, which occurs after the age of thirty years, may be contrasted with a *juvenile* or *benign form*, developing in children, not related to cancer and only half as frequent. The symptoms are usually not so marked and are more stationary; the duration is indefinite, but the general health is not affected. The pathogenesis is unknown.

The *atypical forms* which have been described, can be admitted only conditionally and may possibly be referred to ichthyosiform hyperkeratosis or analogous conditions.

Accidentally Proliferating Dermatoses.—Proliferating lesions may occur in a large number of acute or chronic, generalized or localized cutaneous affections of very variable character. The territory, or the affected tegumentary region is sometimes responsible. It is obvious, although this fact seems to have escaped the attention of most writers, that the peri-anal, peri-genital, inguinal, axillary regions, in short the large folds, and on the other hand the borders of the orifices of the face, the scalp and to a less degree the extremities are especially subject to the occurrence of papillary proliferations. It is also possible that certain infectious agents have a tendency to excite proliferation; several pathogenic protozoa apparently possess this property.

The assumption is, moreover, justified that the papillomatous character which various eruptions may accidentally develop, is referable to a secondary infection or a microbic symbiosis.

Pemphigus Vegetans Gravis.—This pathological type was described in 1876 by J. Neumann, who considered it as a distinct disease. It is characterized by bullæ on the floor of which papillomatous growths very promptly make their appearance. The eruption is situated especially in the groins, in the large articular folds and at the circumference of the mouth. It often begins on the buccal or pharyngeal mucosa, or at the genital organs; sometimes at the border of the finger-nails [or at the anus].

The initial lesion is a bulla, often flaccid and seropurulent from the first, which dries into a crust and may heal locally or spread at the periphery. At the end of five or six days, the floor of some

or most of the bullæ becomes excoriated, begins to proliferate, undergoes a papillomatous change and secretes an offensive pus under a brownish crust. There is a striking resemblance between these lesions and hypertrophic mucous patches. Serpiginous extension and confluence of the lesions give rise to extensive surfaces, mammillated in the center and pustular at the circumference. Healing is followed by brownish and roughened maculæ.

In severe cases, the mouth is lined with very painful diphtheritic erosions, the lips, all the articular folds and the regions where skin rests upon skin, sometimes a considerable portion of the head, trunk and limbs, as well as the mucous membranes, are covered with fetid and painful suppurating vegetating ulcerations. Fever has been repeatedly noted. Death supervenes from cachexia in two to six months, sometimes later.

The histology of the initial bulla is that of genuine pemphigus. In the papillomatous stage, the excrescences, which may reach a height of 6 to 10 millimeters, are covered with a greatly thickened rete Malpighii. In the latter, or in the proliferating papillary body, or between these two tissues, small abscesses are demonstrable, with polynuclear leukocytes and numerous eosinophile cells. Eosinophilia have been demonstrated in the blood and various lesions of the nervous system and the viscera have been found at autopsies. Pemphigus vegetans gravis is observed in both sexes, especially in adults, but it is a rare disease. Its nature is unknown. Its unfavorable course and gloomy prognosis suggest that it is merely a variety of genuine pemphigus, which has become papillomatous through a superadded infection with an undetermined agent. G. Pernet (1907) is undoubtedly right in claiming that pemphigus vegetans is not a definite pathological variety, but is referable to a number of infectious agents among which he states the Bacillus pyocyaneus to be especially common. [The pyocyaneus, however, is extremely common everywhere!]

Pemphigus Vegetans Benignus.—Beside the above described almost invariably fatal form, an eruption is sometimes observed having very similar lesions, but advancing in successive attacks, with preservation of a good general condition and terminating in recovery after a variable length of time. In this benign form, the mouth, lips and large folds are not so much involved; whereas the skin of the extremities and the large surfaces of the trunk (Fig. 85) suffers more.

There is a striking similarity with bromoderma and iododerma vegetans.

The coexistence of polymorphous eruptive lesions and eosinophilia of the blood has often been interpreted as pointing to a proliferative form of Duhring's disease. Non-recurrent cases have on the other hand suggested the idea of erythema bullosum vegetans.

On the whole, the series of forms of pemphigus vegetans presents the same difficulties of classification as the bullous eruptions in general. Some dermatologists simplify the problem by recognizing a dermatitis vegetans capable of assuming every imaginable degree.

Treatment.—The treatment is that of infected erosions of all kinds: prolonged local baths, softening or mildly antiseptic moist dressings, with hypochlorites, permanganate, etc., painting with iodin solution, application of absorbent powders. Arsenic and especially arsenobenzol have yielded some very satisfactory results. Radiotherapy is likewise successful.

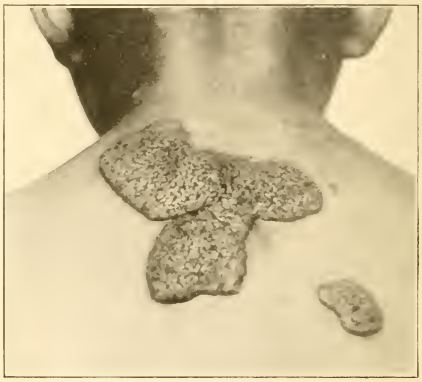


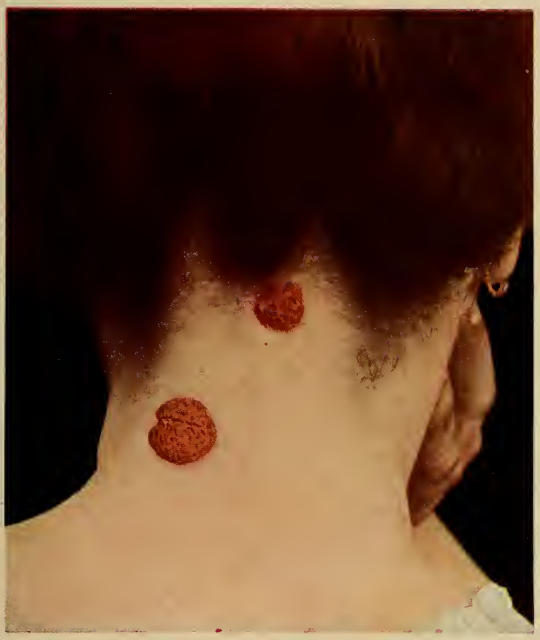
FIG. 85.—Benign pemphigus vegetans; patch of five months' standing in a child aged five years; blood withdrawn from the lesions contained 30 per cent. of eosinophiles.

Proliferating Pyodermatitides.—Under the name of *chronic pustular dermatitis in foci with peripheral extension*, later as pyodermatitis vegetans, Hallopeau (1889–1898) described a form which is pustular from the onset, benign in character, slowly progressive and very protracted, as a variety of Neumann's pemphigus vegetans.

The name of pyodermatitis vegetans might also be applied to the cases in which an impetigo or an infected herpes, etc., give rise to papillomatous proliferations, which is especially apt to occur in the folds. (See Syphiloid Dermatitis Vegetans.)

Papillomatous and Verrucous Elephantiasis.—In all forms of elephantiasis, especially in elephantiasis nostras and still more in the secondary elephantiases, the cutaneous surface, chiefly on the lower part of the legs and on the feet, may become roughened,

PLATE I



Vegetative Syphilides of the Secondary Stage.

The patient presented at the same time vegetative mucous patches upon the lips and under the tongue.

mammillated, covered with pink or whitish proliferations, or with gray or blackish hyperkeratotic verrucosities.

Hypertrophic Lichen Corneus. — This affection, which is very easily recognized after seeing a case, has been described above (p. 140).

Papillomatous Syphilides.—These are either secondary or tertiary. The secondary kind constitute a modification of the lenticular or especially the nummular papules. Generally isolated or not numerous, they occupy the nape of the neck (Plate I), the thorax or the face and assume the shape of distinctly circumscribed, sometimes crusted papillomatous patches, 0.5 cm. thick, from 1 to 4 cm. wide, developing gradually and leaving a dyschromic spot. They are not rare in the nasogenial furrow and at the fold of the chin.

Hypertrophic mucous patches belong entirely under this heading. The syphilitic patches of Legendre are related lesions.

Tertiary proliferating syphilides may develop on a variety of ulcerations, especially on tuberculo-gummous syphilides. In these cases, papillomatous or fungoid papillomatous growths are seen arising from the depths of solutions of continuity, radically altering the appearance of the lesions. This contingency must be kept in mind, for tertiary proliferating syphilides are often confused especially with epitheliomas. They are observed more particularly in hairy regions, the scalp, beard, axilla, pubis and on the lower limbs; they may accompany an elephantiatic state.

Papillomatous and Verrucous Tuberculosis.—The cutaneous tuberculous lesions are proliferative under various conditions. Irregular patches of ulcerative papillomatous tuberculosis may be observed, although not commonly, either at the circumference of the mouth or at the anus, or finally at the vulva, where they are sometimes considered as part of the syndrome of vaginal lupus. Pinkish or papillomatous proliferations arise from the floor or the border of the ulceration. The course is extremely slow.

From time to time, cases of frambesiform tuberculosis are reported, representing large surfaces covered with villous proliferations and interspersed with irregular ulcers and miliary abscesses. A lesion of this kind, which covered almost the entire extent of the thigh and buttock was observed by myself and Brocq.

Tuberculosis Verrucosa.—Tuberculosis verrucosa is more common, and is entitled to a detailed description. It is usually found on the hand, on the fingers or on the wrists; it has a certain predilection for the radial side of the hand and especially for the thumb (Fig. 86); but it is also met with on the elbows, the knees, in the peri-anal region, on the buttocks, the feet, the neck and even on the face. It appears in the form of a single patch, of nummular

dimensions or more extensive, of a round, oval or multilobular configuration, sometimes elongated, following a fold of the skin.

When the focus is small, it is a papillomatous or hyperkeratotic elevation, somewhat resembling a wart; but its base is always surrounded by a red or purplish areola; pressure will occasionally cause a small drop of pus to escape.

In its completely developed state, a patch of verrucous tuberculosis is composed of three zones: at the circumference, a smooth and level *erythematous zone;* next, a more elevated, purplish or brownish, papillomatous *median zone,* interspersed with small adherent crusts or cribriform ulcers, from which droplets of pus can be squeezed out; finally, a *central portion,* which is sometimes cicatricial and depressed, sometimes protuberant and studded with gray or yellowish horny verrucosities, separated by grooves and fissures. The base of the patch is indurated, of fibrous rather than edematous consistence.

FIG. 86.—Tuberculosis verrucosa. After a cast of Baretta's in the Museum of the St. Louis Hospital.

After healing has occurred, the cicatrix is flat and white, or undulating and interspersed with light tracts on a purplish background. It is adherent when the cutaneous lesion was derived from a deep, bony, or glandular focus, etc.

The diagnosis of tuberculosis verrucosa, based on its objective features, its seat, the conditions under which it appears, and its slow course, is usually easy. Examination by biopsy, bacteriological examination and experimental inoculation will serve to confirm it in those cases where syphilis, epithelioma, sporotrichosis, the blastomycoses, etc., cannot be readily excluded.

Anatomical tubercles may be regarded as a verrucous tuberculosis, of small dimensions, but highly virulent.

Discussion of the etiology and treatment of these affections will be found elsewhere in this book (p. 552).

Proliferating or Papillomatous Epithelioma.—This is observed chiefly in the face, especially on the lips or on the mouth and also on the external genital organs.

This papillomatous epithelioma often develops on a basis of senile keratosis or leukoplakia. Surgical excision, as promptly as possible, is indicated.

Mycoses, Sporotrichoses, Etc.—An appearance identical with or closely analogous to that of papillomatous syphilides, papillomatous epithelioma, and especially tuberculosis verrucosa, may be produced by a blastomycotic or sporotrichetic infection. At the present day, when the frequency of the mycotic dermatoses has been established, especially through the work of de Beurmann and Gougerot, it would be a serious error not to take this possibility into consideration, whenever it is plausible. The investigations required for its verification will be discussed elsewhere.

Papillomatous Toxicodermas.—Iododerma papillomatosum may appear in any region of the integument, beginning as a purulent bulla with an inflammatory base, which becomes papillomatous at the center and rapidly extends peripherally. The usually multiple lesions become confluent in sometimes rather extensive patches; their pustular margin is suggestive.

Bromoderma papillomatosum greatly resembles the iodide eruption, but is softer, more fungoid, with less tendency to suppurate.

In both cases, in the absence of definite information, iodin or bromin may be looked for in the urine. It is noteworthy, however, that these eruptions may persist several weeks after the last ingestion of the drug. [In the case of nursing infants the eruption may develop in consequence of a bromide or iodide mixture taken by the nurse.]

PROLIFERATING TROPICAL DERMATOSES.

A group of diseases endemic in tropical countries is characterized by proliferating or ulcero-vegetative eruptions.

Their objective appearance is sometimes typical, but they are nevertheless apt to be confused, especially with tuberculosis and syphilis. In a general way, it may be stated that tuberculosis verrucosa differs by its slow course and the small number of foci which never heal spontaneously; and syphilis, by its polymorphous, protean manifestations, which do not spare the mucosæ and which follow a definite course.

In most cases, a valuable diagnostic indication is furnished by the mode of origin, the disease appearing either in the country where it prevails, or in returning travellers.

Oriental Boil (Biskra Button).—In its developed stage, the Biskra button (p. 648) is a round or oval ulceration, the size of a small coin or several centimeters in diameter, with festooned outlines, concealed under a very adherent yellow-brown crust and bordered by an erythematous margin (Fig. 186).

Under the crust is found a turbid serous fluid, an ulcer with perpendicular eroded ragged margins and a bright-red, granular, mammillated and papillomatous floor. On this floor and around the circumference, purulent yellow points may be found. The base is congested and infiltrated. Lymphangitis and phlebitis are frequent complications. The glands are as a rule enlarged and painful.

Spontaneous cicatrization is slow, requiring several months. The scar which persists is depressed, smooth, colorless or of a dusky red hue, with peripheral pigmentation.

The histological structure is that of a granuloma, with predominance of mononuclears, a few plasmocytes and foci of necrosis. The papillary hypertrophy is considerable, with hyperacanthosis and parakeratosis.

Yaws.—The eruptive lesion in *yaws* or *Frambesia tropica*, in the primary lesion as well as in the generalized eruption, always consists at the onset of a rose-red conical elevation, with a crusted necrotic center; the crust is discharged, the lesion extends and becomes papillomatous (p. 646).

The patches may attain a diameter of 1 to 6 cm.; they are covered with an adherent brown crust, or a fetid secretion in the macerated areas. The proliferations are grayish or pinkish; there is no ulceration or loss of substance. The center has a tendency to collapse; the circumference is often the seat of a bullous elevation. Confluence gives rise to the formation of large patches with polycyclic outlines. The eruption is usually painless.

A spontaneous cure may occur at any stage of development; it manifests itself by the lesions becoming flattened and undergoing absorption. Only pigmented macules are left behind, and, as a rule, no cicatrices.

The *site* of election of the eruptions in yaws is around the natural orifices, at the lips, the nostrils, in the genital regions and in all body folds. But they may be very extensive and profuse, suggesting hypertrophic mucous patches and papulo-crusted syphilides. In the palmar and plantar regions, the proliferations are covered with hyperkeratoses and are very painful. The mucous membranes always remain free.

Histology shows an abundant infiltration of plasmocytes, without epithelioid cells or giant cells; it occupies the papillary body and the papillæ; the latter are enormously hypertrophied and traversed

by dilated vessels. Hyperkeratosis is considerable and parakeratosis is common.

Elsewhere mention will be made of the proliferative papillomatous ulcers of *Boubas*, which are accompanied by similar lesions of the mucous membranes (p. 650).

Ulcerating Granuloma of the Genital Organs (Granuloma Inguinale, Granuloma Venereo, Groin Ulceration).—This is a chronic and contagious, ulcerative or papillomatous dermatosis, venereal in most cases, occurring in all countries, notably in South America, especially in Guiana and Brazil, as well as in New Zealand and the East Indies; a few cases have been reported in England. It attacks adults of both sexes and all races.

Beginning with a nodule on the genital organs, it spreads in the form of an ulcerative granular patch to the groins, the pubis, the perineum and invades the mucosæ. The borders are distinctly marked; the surface is riddled with papillomatous elevations, superficially ulcerated, of a bright red color; it secretes an offensive serous fluid; the center becomes cicatricial. This lesion lasts many years, without causing glandular enlargement or giving rise to cachexia.

Histologically, it is a plasmoma with enormous hypertrophy of the papillæ, analogous to yaws. According to a very recent and excellent contribution by Souza Araujo (Rio de Janeiro, 1917), the causative agent seems to be the germ discovered by Donovan in 1905, which has since been isolated and cultured and is known as *Calymmato-bacterium granulomatis*. This encapsulated bacillus is Gram-negative, takes the Giemsa stain and is abundantly present in the cells of the granuloma and in the secretion. The specific treatment of the disease consists of intravenous injections of tartar emetic.

CHAPTER XIII.

TUBERCLES AND TUBERCULO-ULCERATIVE DERMATOSES.

In the dermatological [as distinguished from the pathological] sense of the word, tubercles are pathological products of the cutis, solid (that is, without fluid contents), circumscribed, rounded, more or less prominent, of slow development, deep and especially damaging to the cutis.

This last named and most essential characteristic involves a frequent tendency of tubercles to ulcerate and almost always to leave a cicatrix in their place, with or without previous ulceration. This fact is expressed in the statement that tubercles do not undergo resolution.

The differentiation of tubercular eruptions from papules, nodules and tumors, must first be clearly understood. Papules are distinguished from tubercles, not so much by their size and prominence, as by their more rapid development and tendency to absorption, a papule leaving no scar behind it after it has healed. Doubtful or intermediate lesions are met with, however, which may be named *papulo-tubercles;* some dermatologists use this term for giant papules.

Nodes and *nodules* are hypodermic newformations, whereas tubercles are dermic. But, although morphologically distinct, tubercles and nodules often result from the same process, justifying the terminology of Besnier, who defines nodules as *hypodermic tubercles.*

Certain tubercles simulate tumors, or approximate these by their size, prominence, confluence in raised and mammillated patches, course, etc. Tubercles, however, after a more or less prolonged duration, have a tendency to become replaced by a cicatrix; tumors, on the contrary, are persistent or indefinitely progressive, although these features are not invariable.

In reality the basis of differentiation is that the name of tumors is given to newgrowths of entirely unknown character, while dermic neoplasms of a known chronic infectious origin are known as tubercles.

The *histological* criterion is decisive in this connection. As will be seen further on (Chapter XXXI), tumors are composed of heterotopic tissues which replace the normal tissue of the affected

region. Tubercles, on the contrary, are formed by collections of inflammatory cells of various types, more or less coherent and extensive, infiltrating the strands of the derma which may have undergone degenerative changes.

The abundance and density of these cellular infiltrations and the degree of preservation of the dermic stroma are variable and account for the more or less marked prominence, firmness or softness of the tubercles. Their constitution also serves to explain their capacity of undergoing absorption, although usually not without atrophy and sclerosis, that is, not without a cicatrix.

In some tubercles, the infiltration has a marked tendency to cellular necrosis, disintegration and dissolution naturally following; the outcome is a limited and often very deep ulceration.

The *differential diagnosis* between a tuberculo-ulcerative process and primary ulcerations rests on its at first neoplastic and secondarily destructive course; furthermore, on the usual persistence of tubercular remnants at the base and in the circumference of the ulcer, indicated by a special hardness in the case of tertiary syphilides or actinomycosis, by a peculiar softness in the case of ulcerative lupus, etc.

Indurated follicular pathological products, whether suppurative like furunculosis and carbuncle, or dry like keloid acne, do not figure in this book among the tubercles, but in the chapter on the folliculoses (Chapter XIX).

TUBERCULO-ULCERATIVE DERMATOSES.

The presence of true tubercles or of a tuberculo-ulcerative lesion having been recognized, it is necessary to think in the first place of the great chronic infectious diseases, *i. e.*, syphilis, tuberculosis and leprosy; secondarily, of the more uncommon mycotic infections, such as sporotrichosis, the blastomycoses and actinomycosis, for which the reader is referred to another chapter (XXVIII); finally, there is a group of dermatoses manifesting itself by non-ulcerative tubercles, the lupoid and analogous cutaneous sarcoids, which are undoubtedly of infectious character and probably belong to the tuberculides, although this has not been positively established.

Only the following types will be described in this connection: (1) The *tubercular syphilides*, (2) the *tubercles of lupus*, (3) the *tubercles of leprosy*, (4) the *cutaneous sarcoids* or *lupoids* and *granuloma annulare*.

Tubercular Syphilides.—From the purely morphological viewpoint, leaving the course entirely out of consideration, the initial symptom of syphilis, the hard chancre, might be said to present the chief attributes of a tubercle.

Genuine tubercular syphilides, however, are symptoms of the tertiary stage.

The syphilitic tubercle constitutes the typical form of a tubercle. It is a dry elevation, brownish or grayish red in color, the average size of a lentil, projecting from 1 to 5 mm., with rounded contours, very hard on palpation, entirely painless. On vitropressure (meaning when the tubercle is compressed under a glass slide), it is seen to be opaque and frequently pigmented.

The subjoined illustration (Fig. 87) conveys an idea of its histological structure.

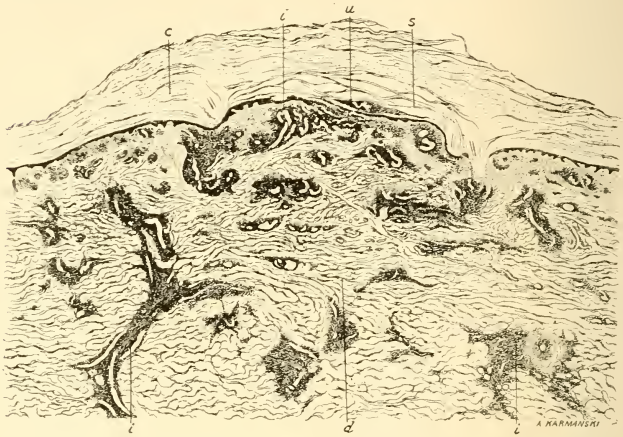


FIG. 87.—Histology of syphilitic tubercle. Tuberculo-squamous tertiary syphilis. The infiltrate (*i*, *i*) is discontinuous, composed of lymphoid cells and plasmocytes without giant cells or epithelioid cells; it forms cuffs which surround the ramifications of the bloodvessels; these are for the most part dilated, inflamed, or sclerotic. Between the infiltrates the dermic tissue (*d*) is thickened, fibrous and sclerotic, which explains the hardness of the lesion. The horny epidermis (*c*) is greatly thickened and coherent; at (*s*) it is split and shows a tendency to desquamation. The Malpighian layer and the papillæ are preserved, except at (*u*), where the inflammation is more active and ulceration is under way. See Pathological Anatomy of the Syphilides, p. 628. × 20.

A tubercular syphilide begins as a single lesion or as a small coherent group of tubercles which spread out, progressing excentrically and multiply within a few weeks; in this way, more or less extensive patches are formed or even larger areas may become involved. Usually, however, the central lesions flatten, fade and undergo sclerotic changes, forming cicatrices even in the complete absence of ulceration, while new lesions make their appearance at the periphery. This centrifugal or serpiginous development

gives rise to circinate forms; these are by no means complete rings, but generally more or less semicircular, having a diameter of 2 to 12 cm, or kidney-shaped and, through confluence, polycyclic configurations (Fig. 88). Their border is marked by a usually interrupted zone of isolated or confluent tubercles.

Occasionally, the eruption of syphilitic tubercles may present this circinate arrangement from the start. In these cases, the central area shows normal skin. When the circination is the result of centrifugal extension of the process, its center is, on the contrary, often depressed, slightly adherent and not readily folded; it may be distinctly sclerotic or interspersed with cicatricial stars and streaks; its color is earthy rather than white, often mottled with purplish or

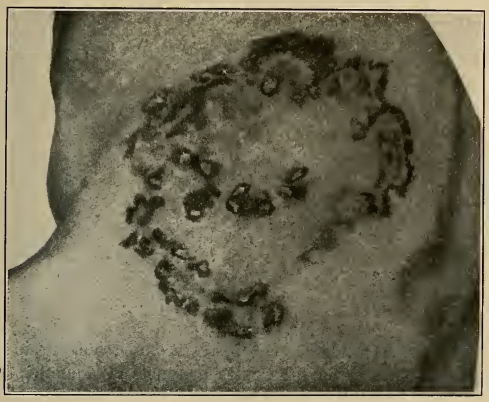


FIG. 88.—Tuberculo-circinate syphilides of the left hip.

brownish shades. The reappearance of tubercles on the cicatrix is rare in the tuberculo-circinate syphilides; whereas, on the contrary, this occurrence is frequent in lupus serpiginosus.

After recovery, the tubercular border has disappeared, but the persisting cicatrix may still present a very characteristic appearance.

Various forms of tubercular tertiary syphilides may be described:

The tuberculo-squamous form, with grayish, more or less abundant, adherent scales, is very common, more so than the tuberculo-psoriatiform variety, with abundant nacreous scales and barely perceptible infiltration; it is distinguished from psoriasis by the small number of the patches, their configuration [and location] and sometimes by the presence of cicatrices.

When the tubercles become eroded and covered with an adherent dirty brownish crust, the outcome is the tuberculo-crusted form, which is connected by imperceptible transition with the tuberculo-ulcerative form. In the latter, the crusts are found to cover round, perpendicular-walled ulcerations containing a sanious pus.

These various forms are usually circinate. There also occurs, notably on the nose, the forehead and chin, a *superficial tubercular syphilide*, composed of large, smooth, dusky red tubercles, which may be agminated or sometimes confluent in an infiltrated patch. The latter is apt very closely to simulate rosacea in the stage of rhinophyma; when it appears that the center has a tendency to become sclerotic and depressed, this fact furnishes a valuable indication in favor of syphilis; but a careful investigation may be necessary.

Certain tertiary syphilitic ulcers, characterized by their indurated base, sharp outlines and their course, may be considered as tuberculo-ulcerative syphilides with indistinct and primarily confluent tubercles (Fig. 98). The same remark applies to chancriform syphiloma.

The *diagnosis* of tubercular syphilides can very often be based upon their objective appearance; not uncommonly, they put the alert observer on the track of an undetected long-standing syphilis. In other cases, the diagnosis will be confirmed by the complete examination of the patient, the anamnesis and serodiagnosis, with examination by biopsy if necessary.

Specific *treatment* will cure these lesions in from two to four weeks.

Lupus Tubercles.—Lupus vulgaris is one of the most attenuated forms of bacillary tuberculosis of the skin. Its characteristic lesion consists of a tubercle possessing certain special features.

In order to avoid the ambiguousness resulting from the double meaning of the word tubercle—bacillary tubercle and dermatological tubercle—it has been recommended to designate the eruptive lesion of lupus as *lupoma*.

All cases of lupus begin as a minute lupoma, which grows to the size of a pin-head, then of a lentil, while other similar lesions more or less rapidly originate in its immediate circumference or in its vicinity. Sometimes, lupus begins in two or three distinct foci, or exceptionally in the form of an extensive eruption. Lupomas may accordingly be found isolated or conglomerated in patches around which a few aberrant lesions are apt to appear.

Typical *lupomas* are rounded tubercles, the size of a pin-head to that of a large pea, more or less prominent, or on the contrary, perfectly level with the normal skin; their color is a yellowish, sometimes purplish or dusky red; their surface is smooth and shining, or scaly, eroded, crusted or ulcerous; their consistence is

remarkably soft, compressible, velvety; they are easily penetrated with sharp instruments; they are often tender on touch.

When a lupus tubercle is subjected to vitropressure or diascopy, to drive out the blood, its tissue is seen through the glass slide to be of a translucid deep yellow color, comparable to barley sugar or apple jelly, distinctly outlined from the creamy white surface presented by the normal derma. This pathognomonic transparency of the compressed lupoma, which is due to the local disappearance of the elastic and connective-tissue network (Fig. 89), is easily distinguished from the opaque coloration shown under the same conditions by a pigmentary spot or a soft verrucous nevus.

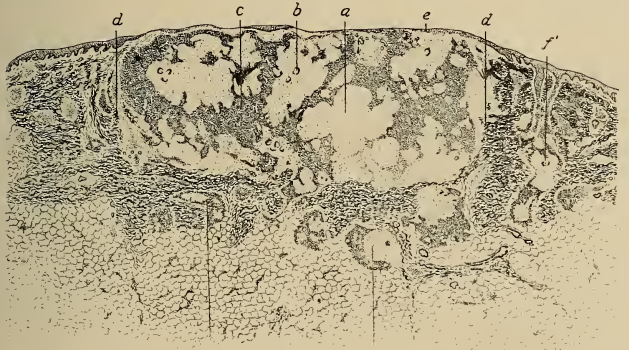


FIG. 89.—Histology of lupus tubercle. Nodular lupoma of the cheek. Stained with acid orcein and polychrome blue. The tuberculous newgrowth occupies the entire thickness of the derma and results from an agglomeration of tubercles such as those seen in Fig. 93. *a*, collection of epithelioid cells; *b*, giant cells; *c*, infiltrate of lymphocytes and plasmocytes forming a finely granular network; *d*, *d*, elastic and connective-tissue framework of the derma, plainly seen to be interrupted at the level of the tubercle (accounting for the softness and transparency of the latter); *e*, tense thin epidermis almost eroded on the surface of the lupoma; *f*, *f*, rows of lupous infiltration, seen extending into the hypoderm or (*f'*) around a hair follicle. × 15.

Lupoma, as a rule, has a tendency to persist indefinitely, gradually extending at its periphery and becoming confluent with neighboring lesions. The duration of its course extends over months, years or decades. However, even in the absence of all therapeutic intervention, two contingencies may arise:

Some cases, so-called *resolving* lupus, will heal spontaneously and become cicatrized, the interstitial sclerosis more or less completely strangling and extinguishing the cellular newforma-

tion. This spontaneous cicatrization usually takes place in the center of the patch, while the border continues to proliferate (Fig. 168); lupomas are often seen to persist or reappear in the cicatrix, which is smooth, white, pearly and more or less flexible. This last-named fact is of positive diagnostic value in the differentiation from circinate and serpiginous syphilides. The treatment of lupus by scarification, punctate cauterization, light- and radiumtherapy, etc., aims at stimulating and favoring the process of cicatrization, which is the natural mode of cure.

On the other hand, the lupoma may undergo necrosis and ulceration. The tendency to central necrosis which belongs to all processes caused by the tubercle bacillus is relatively much less marked in lupus than in the other forms of cutaneous tuberculosis. In some cases, however, it may become more pronounced, giving rise to restricted, rarely extensive or even enormous destruction of tissue; the latter being characteristic of *lupus exedens* and *vorax*. The features of lupus ulcerations will be discussed elsewhere in this book.

Tubercles of Leprosy.—The tubercles of leprosy, or *lepromas*, originate either on erythematous pigmented spots or on the healthy skin. They may appear separately and insidiously, or in crops of numerous lesions, distributed with a certain symmetry on the face (Fig. 175), on the limbs, or in any region of the body.

Their usual appearance is as follows: Their size varies from that of a hemp seed to that of a large almond; their shape is hemispherical; their elevation is variable; the color is a dull pink, or purplish, or brownish; their surface is smooth, always hairless, sometimes oily or scaly; their consistence is firm at the onset, but rather softish and shrunken often after the lapse of some time.

Their principal properties are: (1) They are almost invariably anesthetic toward pricks or burns, sometimes after a temporary stage of hyperesthesia; (2) they are histologically composed of collections of lepra cells, teeming with Hansen's bacilli.

Lepromas may become confluent in lobulated tumors, or they may spread out in patches of variable extent, moderately prominent, with a smooth or irregular surface interspersed with telangiectases, etc.; when they occur in patches they are designated as *leprous infiltrations* or *surface lepromas*.

The duration of leprosy tubercles is usually very long, but follows no rule. They may disappear through absorption, leaving a white or pigmented cicatrix; this scar of leprosy infiltrations bears the name of *leprous morphea*.

They may also undergo suppuration and ulceration; a primarily superficial erosion increasing in depth and growing into an irregular deeply excavated ulcer, with swollen margins, and a sanious floor, sometimes becoming phagedenic and mutilating.

Lepromata are characteristic of the tubercular or generalized cutaneous form of leprosy and are also encountered in the mixed form (Chapter XXVII).

Local *treatment* of leprosy tubercles yields very satisfactory results. They may be treated with salves or plasters containing strong reducing agents, or preferably by cauterization with the galvanocautery, which leads to their prompt subsidence. [High-frequency and radiotherapy are valuable measures, but it need hardly be said that local treatment of a few nodules has little or no effect on the course of the disease.]

Cutaneous Sarcoids or Lupoids.—The eruption, which was first described by Boeck (1899) under the name of *multiple benign cutaneous sarcoid*, then as *benign lupoid*, manifests itself in the form of lenticular or larger tubercles, which never become ulcerated.

General remarks on the sarcoids will be found further on and are applicable to the lupoids, which in the main represent merely a superficial dermic type of sarcoid, with lesions of small dimensions, affecting more particularly the face and the upper limbs.

A distinction is made between two principal forms:

1. **Disseminated Miliary Lupoid.**—The eruption consists of hemispherical elevations, the size of a millet seed to that of a large pea, of a pinkish, then livid and finally brownish color, with a smooth or very slightly scaly surface, of semi-solid consistence. On vitropressure (compression under a glass slide) their tissue is less transparent than that of a lupoma and often seems to be made up of separate granules.

The eruption is symmetrical and situated on the face, the shoulders, the wrists (Fig. 90) and in general on the extensor surface of the upper limbs; more rarely, lesions are noted on the scalp, the back, or the lower limbs. It comes on in a few weeks, but increases during months and years through growth and multiplication of the lesions. In the course of time, the latter become flattened, spreading out in nummular, sometimes marginated, spots and finally become obliterated, leaving an atrophic often not very noticeable cicatrix. The lesions are accordingly tubercles, in the dermatological sense of the term. They never ulcerate, in notable contradistinction to the lesions of acnitis.

The duration of the disease when left to itself is very variable, from five to ten years or longer. It is observed much more frequently in women, between the ages of fifteen and forty years, than in men. The lymphatic glands are sometimes enlarged. In a considerable number of the cases, the patients are evidently suffering from glandular or visceral tuberculosis.

The *histology* of lupoid tubercles is characteristic. The derma is found to contain large lobulated or branching collections formed

chiefly of epithelioid cells, lymphocytes and a few rare giant cells; these collections are separated by connective-tissue strands, in which practically no trace of inflammation is demonstrable.

Several modes of *treatment* have proved efficient, notably intramuscular injections of calomel, tuberculin injections and, according to Boeck, arsenic medication; the best method of treatment actually consists in combining novarsenobenzol injections with tuberculin, injected in very small doses. It goes without saying that the hygienic condition of the patients must be supervised.

FIG. 90.—Disseminated miliary lupoid. Multiple benign cutaneous sarcoid of Boeck.

2. **Nodular Lupoid.**—Nodular lupoid and lupoid in patches (large nodular form of Boeck) is less well known. It consists of hemispherical, purplish or brownish red elevations, the average size of half a hazel-nut, or in other cases of irregularly outlined soft disks; these lesions, to the number of two or three up to ten or more, occupy the forehead, the nose, the shoulders, the elbows, the neighborhood of the knees, etc.

Confusion is possible with lupus, superficial tubercular syphilides,

or leprosy infiltration. Their histology is related to that of lupus, but inoculation of their tissue into guinea-pigs does not produce tuberculosis. The tuberculin reaction is not constant.

I have thought that these two forms of lupoid which I have seen coexisting with tuberculides or with glandular and pulmonary tuberculosis, were tuberculides; this is very probable, but not certain. Görgen Schaumann (January, 1917) interprets the condition as a special benign infectious lymphogranuloma, of the same character as lupus pernio.

Granuloma Annulare.—Undoubtedly identical with the *ringed eruption* of Colcott Fox, the *lichen annularis* of Galloway, the *sarcoid tumors* of Rasch, the *nodular* and *circinate neoplasias* of Brocq, the *granuloma annulare* of Radcliffe Crocker is characterized

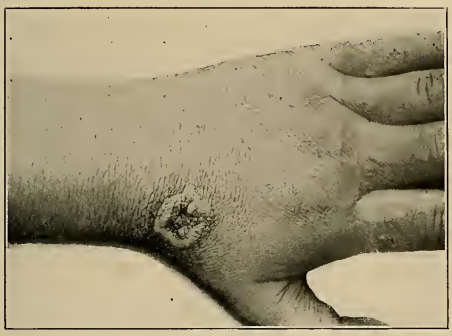


Fig. 91.—Granuloma annulare. The patient, a girl of three years, presented seven other similar lesions upon the limbs.

by elevations or tubercles grouped in rings. At the onset, a firm, smooth nodule the size of a small pea develops rather rapidly; by eccentric growths, or by accession of other nodules, this becomes transformed into a ring (Fig. 91); the color is a pale or dull pink; there is absolute painlessness. The course is very slow, lasting months or years; the lesions never ulcerate and no scar is left after healing.

This affection, which is rare, is observed in children or adults of both sexes and occupies more particularly the hands, the finger-joints and the wrists as well as the ankles, but also the elbows, the knees, the buttocks, the nape of the neck, exceptionally the face. As a rule, the lesions are few in number. Histology shows a perivascular infiltration of lymphoid and epitheloid cells, deeply situated in the corium; the epidermis is intact.

Granuloma annulare in many respects approximates the sarcoids and the lupoids. Graham Little, in his very complete study of the subject (1908) is inclined to consider it as a tuberculide. It is treated with reducing plasters and the same general medication as the sarcoids.

The *erythema elevatum diutinum* of R. Crocker is probably merely a disseminated or grouped but not annular variety of the same affection.

CHAPTER XIV.

NODES AND NODULES.

NODULAR DERMATOSES.

Under the name of nodes I comprise all circumscribed indurations of the hypoderm, of whatever character. Nodes may attain the size of an egg, or larger; nodules in my nomenclature are those having the average size of a pea; nodosities are those having intermediate dimensions.

Nodes constitute an elementary dermatological form, a symptom comparable to an eruptive lesion. They play a part which must not be underrated in the pathology of the skin. In my opinion, they must of necessity figure in the morphology of the dermatoses.

The hyperderm is closely related to the corium by the continuity of the connective-tissue and elastic fibers passing from one to the other as well as by its blood and lymph vessels; furthermore, the bulbs of the largest hairs and the glomeruli of the large sweat glands are imbedded in the hypoderm.

On account of this inter-relation of tissues and vascular supply, pathological processes are very apt to invade the two layers together or successively, so that many pathological products are dermo-hypodermic.

The group of nodes, formerly designated as phyma (French: noudures; German: Knollen [or Knoten]), is usually subdivided in France and classed in part under subcutaneous tubercles, in part with gummas or even with tumors, etc.

It seems to me advantageous to devote a special chapter to their discussion.

Nodes or nodosities may be subdivided according [1] to their volume, their consistence, their more or less distinct limitation; but the diagnostic value of these features is insignificant; [2] according to their purely hypodermic or dermo-hyperdermic seat, but this seat may vary in the course of their development; [3] according to their inflammatory, embolic, or neoplastic pathogenesis; this is not easy to determine clinically. It is preferable to base a classification on their *course*.

From this viewpoint, three forms can be distinguished:

A. *Acute nodes* and *nodosities* of sudden onset, are of ephemeral or not very prolonged duration, from one to fifteen days, for instance, and always terminate by resolution, without suppuration.

In this acute form, the size of the products varies from that of a pea to that of a hen's egg; their consistence is resistant or edematous; their boundaries are indistinct, in the sense of their occupying simultaneously the subcutis and the cutis, with somewhat diffuse outlines; the skin is usually congested on their surface; they are painful to touch. Their abrupt appearance, in certain regions of predilection, their distinctly inflammatory character, their tendency to resolution are suggestive of their resulting from septic emboli of low virulence.

Among the dermatoses in which acute nodes are met with, I shall describe *erythema nodosum* and *rheumatic nodosities*. Giant urticaria might likewise figure in this connection.

B. *Subacute nodes* and *nodosities* develop insidiously and last from a fortnight to several months, or even a number of years. Their volume usually varies from that of a hazel-nut to that of a green almond; their consistence and the appearance of the skin are modified according to their course of development; they are only slightly painful. Their pathological anatomy shows them to be derived from a subacute inflammatory process, frequently having a venous or arterial point of origin and generally of specific character. These cases accordingly represent syphilitic, tuberculous, leprous, mycotic, or analogous newformations.

In this subacute form it is necessary to distinguish a group of nodosities with a well-marked tendency to softening and ulceration: these are designated as *gummas*.[1]

Under the name of *gummas* will be accordingly discussed, first in order: *Syphilitic gummas, tuberculous gummas* and finally *mycotic gummas*, a more recent addition to our knowledge.

Another paragraph will be devoted to a discussion of *subacute non-gummous nodes*. Their course and duration are very variable; some of them never become softened and ulcerated, others exceptionally so. Under this heading I group: the *nodular syphilides*, the *sarcoids*, which are probably *hypodermic tuberculides*, and the *hypodermic lepromas*.

C. If it were desirable to establish a group of *chronic* nodes or *nodosities*, persisting indefinitely, one might do so at the expense of the *hypodermic tumors*, which exist in all varieties of size and consistence. Among such tumors as are sometimes capable of giving rise to serious diagnostic difficulties, the following may be mentioned.

Certain subcutaneous hard fibromas; many molluscoid nevi and fibroma molluscum, such as those of Recklinghausen's disease; a

[1] The student's attention must be called to the fact that the French employ the term Gomme (gumma) in a wider sense than is customary in English. The significance of the term is obvious from the text.

considerable number of cysts; a few deep epitheliomas, cylindromas and metastatic carcinomas; lipomas, myomas, calcareous tumors, deep angiomas and many sarcomas (Chapter XXXI).

ACUTE NODULAR DERMATOSES.

Erythema Nodosum.—This affection is usually considered as a simple variety of polymorphous erythema; but it certainly represents the most individualized type of the disease.

Erythema nodosum is characterized by an eruption of nodosities or nodes, dermo-hypodermic from the start, rounded or oval, from the size of a bean to that of a large nut, of pinkish, carmine-red or purple color, rather prominent, not very distinctly outlined, painful to touch. They appear in a few hours, often with general disturbances consisting of fever, malaise, prostration, rheumatoid pains or actual arthritides; their number varies from a few lesions to about thirty; they are found scattered, more rarely arranged in groups on both legs, on the back of the feet, the thighs and sometimes on the forearms, the arms and the buttocks. The bluish color of the lesions, which often does not disappear on pressure, is due to interstitial hemorrhage; it undergoes the color variation of blood in course of absorption and has given the name of *contusiform dermatitis* to this affection.

The topographical distribution of the eruption, which is practically always chiefly localized on the legs, its spontaneous onset, its course and its duration which as a rule does not exceed two or three weeks, permit it to be easily distinguished from traumatic contusions, gummas, sarcomas, etc.

The *etiology* of this eruption agrees in indefiniteness with that of polymorphous erythema; sometimes it becomes associated, moreover, with other manifestations of this erythema. It may be observed in the course of septicemias or toxicodermias, notably in iodism. Its frequency has been commented upon in venereal clinics, among patients suffering from gonorrhea or syphilis. Mauriac and others, without sufficient reason, have actually admitted the existence of a syphilitic erythema nodosum. Formerly, all nodular erythemas were regarded as rheumatic. There is now a tendency to hold *tuberculosis* especially responsible; in support of this association, the practically general significance of which he had asserted since 1907, Landouzy recently reported a case in which, with his collaborators, he discovered a Koch's bacillus in a blood-vessel and successfully tuberculized a guinea-pig with a portion of the same nodule removed for biopsy.

The *pathological anatomy* of the nodes of erythema reveals an acute inflammation of ordinary type, with extravasation of red

corpuscles and a perivascular infiltration of round cells between the adipose lobules; these lesions necessarily suggest an origin from microbic emboli, placing them in the class of "phlebitides of the cutaneous veins," studied by Philippson. Although it seems to be an established fact that a transitory tubercular bacillemia is responsible in a certain number of cases of nodular erythema, it must not be overlooked that the immediate prognosis of this affection is in the main always favorable and that the prognostic significance of this blood infection must not be overrated.

The *treatment* is that of polymorphous erythema; rest is especially necessary.

Rheumatic Nodosities.—The nodosities known under this name must be approximated to erythema nodosum. Several types have been described, all equally uncommon. In some arthritics suffering from subacute rheumatism and intestinal dyspepsia, I have observed the appearance of crops of nodules the size of a pea, subcutaneous and without change of color of the skin, or intradermic and rose-colored, of firm consistence, very painful on palpation. They were situated especially around the knees, at the wrists, the shoulders, etc., and lasted from twenty-four to forty-eight hours.

Féréol observed non-erythematous, very transitory, painless nodosities, adherent to the skin, located especially on the forehead, but always in small numbers.

The type pointed out by Meynet concerned deeply adherent, hard, elastic, or edematous subcutaneous nodosities, without reddening of the skin and slightly painful. Their volume varies from that of a pea to that of a walnut. The crops consist of a fairly considerable number of lesions, situated especially over bony protuberances, more particularly on the skull. Their duration is from a few days to several weeks. Confusion must be guarded against with gummas or with tumors.

Possibly these clinical forms are varieties of the same species, but it is more probable that this syndrome expresses the result of benign microbic embolisms of variable character.

The *treatment* consists in the administration of salicylates and calcium salts.

GUMMAS.

Gummas are nodular pathological products, of infectious character and subacute behavior, the course of which comprises four stages: (1) Development; (2) softening; (3) ulceration and evacuation; (4) repair. They are situated in the *subcutis*, where they originate primarily around the blood and lymph vessels of this tissue; sometimes, they may reach it secondarily to a process of the same character developing in a subjacent organ, periosteum, gland, etc.

Many authors, who regard the essential feature of a gumma as consisting of its special evolution, rather than of its hypodermic seat, describe *dermic gummas*, developing in the cutis; this term is interchangeable with tubercles. All that need be said is that tubercles occasionally follow an evolution like that of gummas.

Hypodermic gummas, or true gummas, which are alone discussed here, are syphilitic, tuberculous, or mycotic.

Syphilitic Gummas.—These represent typical lesions of this group. The gumma begins as a limited induration of the hypoderm, perceptible to the touch before it becomes visible; it gradually enlarges, setting up around it an inflammatory reaction, first raises and then invades the derma and finally almost invariably opens externally through a crater-shaped ulceration.

In the developed stage, the nodule is of firm consistence, the size of a pea to that of a walnut, indolent and movable. The mass becomes pasty, then fluctuating, superficially and sometimes deeply adherent; the skin becomes reddened and thinned. Up to this time, absorption may occur without leaving a cicatrix, both spontaneously and especially with the help of specific treatment.

More frequently the skin, raised and eroded from below, becomes perforated on top of the protuberance; ulceration occurs and leads to the escape of a turbid or purulent yellowish ropy [gummy] fluid. The orifice enlarges, remaining round, of nummular dimensions; its borders are thin, red, overhanging or perpendicular; the cavity is deep, cup-shaped; the floor is uneven, roughened, covered with a yellowish-white substance, a sort of core, which is gradually eliminated with the discharge; the base is of pasty consistence and tender on pressure. The corresponding lymph nodes are not enlarged.

In the stage of retrogression, the granulating floor becomes raised to the level of the borders where epidermatization begins, concentrically retracting and finally cicatrizing the ulcer after the infiltration has disappeared. The redness is replaced by a persistent pigmentation which surrounds the white and smooth cicatrix.

This evolution extends over a space of time from fifteen to forty-five days.

Syphilitic gummas are tertiary manifestations. Sometimes they are precocious, appearing in the second half-year after infection, and in this case they are called secondo-tertiary, according to Fournier's expression. Although especially frequent in the third and fourth year, they may still occur after ten or fifteen years or more.

They may be situated anywhere, rather commonly on the forehead, on the scalp, at the lips, the genitals; they are often multiple or develop in close succession to the number of from two to ten or thereabouts, rarely more.

Gummas of the tongue are rather rare. They appear in the form of one or several hard intramuscular nodes the size of cherry pits on hazel-nuts, studding the tongue and causing more inconvenience than actual pain. They open as large ampullary or crater-shaped ulcers, which heal leaving a very minute cicatrix. It is important to distinguish them from the *sclero-gummous ulcers.*

Gummas of the palatine velum are rarely discovered before they ulcerate. They give rise to merely slight inconvenience during a week or two, then perforation of the palate suddenly occurs without pain, often during a meal, manifesting itself by the return of liquids through the nasal fossæ and by nasal speech. The gravity of this complication is due to these functional disturbances, which are persistent, cicatrization taking place without closing the perforation. Recourse must be had to a [plastic] operation or a prosthetic apparatus. Perforation of the palatine velum has long been considered as a reliable stigma of syphilis or congenital syphilis. It has been reported under other conditions, notably after scarlatina (?).

In the *treatment* of gummas, incision of the swelling has no advantages and is only too often practised erroneously. Even total excision, with careful suturing of the wound, would be preferable; but syphilitic gummas, even after softening has occurred, heal as a rule remarkedly well under specific treatment in all its forms. The importance which was formerly attached to iodin in the treatment of gummas, is undoubtedly due to the confusion which prevailed between syphilitic gummas and those now known to be of mycotic character. Very dilute local injections of soluble mercurial salts are very efficient and may be utilized.

Tuberculous Gummas—also known as *scrofulo-tubercular gummas,* or abroad as *scrofuloderma*—differ from the preceding by a few details in their objective appearance and by their more gradual, less continuous evolution.

The hypodermic nodosity which marks the onset of a tuberculous gumma, is sometimes adherent from the start to the deep aspect of the derma, which rather early presents a lavender, purplish, or livid hue. Softening often begins superficially without affecting the entire indurated mass; it may be absent or indefinitely delayed. The fluid is sanious or even serous, turbid and mixed with blood. Ulceration is frequently of an irregular configuration, with soft, detached, purplish borders; the cavity is irregular, sinuous, sometimes sending fistulous tracts in different directions; it is lined with gray or purplish filamentous detritus; its walls are softened or locally indurated.

It is not uncommon for the multiple orifices of the same gumma, or of neighboring gummas united in a *gummous infiltration,* to intercommunicate by sinuous tracts which lead into the same cavity;

they are separated by bridges which may persist even after cicatrization or may rupture as the result of erosion.

Tuberculous gummous ulcers accordingly often have sinuous and ragged, undermined, pale or dark purple borders and an extremely irregular floor. The discharge is very variable, distinctly purulent, or serous and mixed with blood. The crusts possess corresponding features.

These ulcers may invade the tendon sheaths, the joints and the bones, from which they may also be derived. They are sometimes transformed into fungoid tuberculosis or lupus. When they heal, the cicatrix is usually irregular, often adherent in the depth of the tissue, honey-combed with fibrous islands, dentated at the periphery or undermined by fistulous tracts which pass under cicatricial bands. The borders remain for a long time purplish, later becoming pigmented.

The course is slow, with exacerbations and remissions, lasting several months or sometimes years.

Scrofulo-tubercular gummas are frequently but not invariably encountered in individuals whose general condition is unfavorable, who suffer from visceral tuberculosis or especially from bony or glandular tuberculosis. Children and youthful individuals are the most susceptible. They number from one or two to about ten, rarely more lesions presenting different stages; they occupy especially the extremities or the neck, sometimes the trunk, but often the face. They are disseminated, more particularly in children and youthful individuals; or they may show a regional distribution, especially when they are consecutive to a deep focus and, in these cases, assume a lymphangitic type.

Tuberculous gummous lymphangitis, practically limited to the extremities, originates from a lesion of the extremities, usually an anatomical tubercle, tuberculosis verrucosa, spina ventosa, or tuberculous caries of the bones. Arranged along the tract of the lymphatics coming from this primary focus, a series of several gummas begins to develop. A lymphatic strand may occasionally be felt which unites these secondary foci. The corresponding glands are usually indurated, sometimes suppurating or fistulized. The duration of this trouble is indefinite; it may be prolonged until death occurs from vertebral or visceral tuberculosis and from cachexia.

The *pathological anatomy* of tuberculous gummas will be discussed elsewhere in this book. It will be seen that they usually contain Koch's bacilli, although in moderate numbers, and that their tissue causes tuberculosis in guinea-pigs inoculated with it. However, clinically identical products are also met with whose scrapings and sections contain no bacilli and whose inoculation into

animals remains negative. This justifies the conclusion that the clinical picture of scrofulo-tuberculous gumma may be produced by hypodermic tuberculides.

The same picture may, moreover, be very closely simulated by mycotic gummas; a thorough examination of the patient in regard to his antecedents and the coexistence of internal tuberculous lesions, is imperative for the diagnosis, although in many cases only the laboratory findings are capable of confirming it.

Mycotic Gummas.—Several infections through fungi may give rise to genuine gummas. **Sporotrichosis** is the best known of these and also the most common. *Sporotrichotic gummas* are sometimes disseminated, sometimes lymphangitic and regionally distributed. When disseminated, they may be found anywhere, in variable number; they are indolent and develop in six to eight weeks. Firm at first, then soft, they persist for more or less time in this condition, then undergoing ulceration and evacuation. Their liquid contents are at first viscid and gummous, later on purulent and thickened. The ulcer may be covered by a crust, of ecthymatous appearance, or again very deep, of rounded or irregular circumference, with undermined margins; sometimes, the floor is covered with granulation or even large proliferations. This ulcer remains stationary a long time, unless treatment intervenes (p. 605).

Sporotrichotic gummous lymphangitis consists of a series of gummas developing in the centripetal lymphatic territory of the initial focus (Fig. 180), which may be dermic or hypodermic, or bony, etc. A moniliform strand may connect the lesions. The corresponding glands are often, but not always swollen and enlarged.

Actinomycosis, in its primary cutaneous form, often has the appearance of a hypodermic nodule, with a pinkish surface, hard and practically painless, which softens at its center and ulcerates, but from which a sanguinolent discharge escapes instead of pus; the orifice remains fistulous. Neighboring nodules form and become agglomerated with the first. The lymph glands remain intact. The site of the lesions is most commonly the cervico-facial region (p. 599).

Some **blastomycoses** may give rise to gummas. This is particularly true for the Buschke type, of which a few rare observations have been recorded; it is a febrile infection, seriously interfering with the general health. The origin, as a rule, is in the osseous system whence there follow multiple disseminated gummas. [It would seem probable that the point of entry for the organisms is an insignificant cutaneous lesion, though a periosteal swelling may be the first lesion to attract attention] (p. 603).

Mycetoma, or *Madura foot,* is a local mycosis characterized by nodosities which become bullous on their surface, then softened and

ulcerated, accordingly representing true gummas; their agglomeration may result in a monstrous deformity.

In "*pian-bois*," a Leishmaniosis, I have observed a sort of gumma with a lymphangitic distribution.

For the *diagnosis* and *treatment* of these mycotic gummas, the reader is referred to Chapter XXVIII.

[All the forms of cutaneous and subcutaneous tuberculosis are relatively rare in America and blastomycosis seems to be more common than sporotrichosis while the converse is true in France and Germany.]

SUBACUTE NON-GUMMOUS NODES.

Nodular Syphilides.—This name, as well as that of nodular syphilitic phlebitis, is applied to hypodermic nodules which sometimes occur in the course of the secondary manifestations of severe syphilis, associated in most cases with a profuse eruption of lenticular papules.

The nodules are hard, distinctly outlined, movable under the skin and on the underlying tissues, round, spindle-shaped or flattened, the size of a large pea to that of an almond; the skin on their surface is normal or slightly reddened. They are painless when left alone, but slightly tender on pressure. They are situated in variable numbers, from a dozen to twenty at most, on the extremities, rarely elsewhere.

Their size, their topography, their number, their free mobility, and especially their course, serve to distinguish them from the nodosities of erythema nodosum and syphilitic gummas.

In a case, observed with Civatte, the histological examination enabled us to recognize distinctly the presence of a syphilitic newformation (of the same type as the lenticular papule) which developed in the wall of the subcutaneous veins giving rise to thrombophlebitis.

The evolution of these nodular phlebitides is rather slow. They have practically no tendency to undergo softening and ulceration, but in the absence of treatment will persist for many weeks without change. Under the influence of specific treatment, they very promptly disappear.

Leprous Nodosities.—These products, known also as hypodermic lepromas, often accompany the dermic leprous tubercles in moderate number. In more interesting but less common cases, the nodosities occur without tubercles, coincidently with erythemato-pigmentary leprides. They can be detected only after careful search; as stated by Leloir, they may be felt rather than seen, for they barely raise the skin.

Hypodermic lepromas are situated chiefly on the buttocks, the back, the external aspect of the extremities, on the face, and very frequently on the ear-lobules, which appear as if stuffed with shot.

They are circumscribed, rounded or oval, the size of a pea to that of a walnut, sometimes conglomerated, or they may be diffuse (leprous infiltrations) in slightly raised, flat or uneven patches.

At the onset, the circumscribed nodules are firm and elastic and at first movable; later on they become softened and adherent to the skin, which turns red on the surface. Nodules and infiltrations may ulcerate, thus becoming entitled to the name of *leprous gummas* or *gummous infiltrations of leprosy;* they contain a grumous pus very rich in Hansen's bacilli. They often seem to persist indefinitely, or they undergo absorption, leaving a sclerotic or even keloid cicatrix, or no trace of their former presence.

SARCOIDS.

This term was devised by Kaposi to designate newformations resembling sarcoma. Boeck (of Christiania) described under the name of *multiple benign cutaneous sarcoids*, eruptions of tubercles resembling lupus; more recently, he called them *lupoids*. Other newformations have also been designated under the name of sarcoids. In a report to the XVI International Medical Congress (1909) I pointed out that until the etiology and real character of these new-formations are elucidated, the denomination *sarcoids* is indispensable.

It designates a group of connective-tissue newgrowths which manifest themselves clinically in the form of nodes, nodosities, nodules or tubercles, usually multiple, painless, of slow or even chronic, but not unlimited development. They are benign in the sense that they have no tendency toward softening and ulceration, do not recur locally after removal, do not give rise to visceral metastases and do not notably affect the general health. They are subject to retrogression, either spontaneously or under the influence of arsenical agents, mercury, tuberculin, etc.

Anatomically, these sarcoids are made up of cellular collections or infiltrations often of a tuberculoid structure, insinuated into the framework of the derma or the hypoderm—which distinguishes them from the sarcomas, whose constituents are arranged in coherent and homogeneous masses.

The following pathological types come under this definition:

1. **Cutaneous Sarcoids.**—Cutaneous sarcoids of Boeck, or *lupoids*. Their intradermic seat and their small dimensions have led me to group them among the tubercles (p. 259).

2. **Hypodermic Sarcoids.**—As described by me with Roussy (1904 to 1906), these are painless subacute or chronic newformations, the size of a bean to that of a walnut, or larger; they often become confluent in large uneven patches and nodular strands. The skin over them is raised, of normal, lavender, or dull red color. At first sight, they might be mistaken for tumors, but they have no unlimited extension. They are observed in adults of both sexes; their duration is indefinite.

These sarcoids are generally grouped in certain regions, and have a tendency to a symmetrical arrangement. I know of two seats of predilection: on the one hand, the costal region, the flanks, and the scapular region, on the other, the anterior aspect of the thighs, and the lower portion of the abdomen. However, they are also met with elsewhere, on the arms, the forearms and even on the scalp. This type is relatively rare.

These sarcoids must not be confused with tumors of various kinds of the thoracic wall, nor with axillary, cervical or other adenopathies. On the thighs they may be very closely simulated by nodosities due to the injection of camphorated oil, or other hypodermic injections, which sometimes persist indefinitely. The appearance of genuine sarcoids, however, seems to be absolutely spontaneous. The paraffinomas will be mentioned further on.

3. **Disseminated Nodular Sarcoids.**—These are nodes or nodules which develop successively in crops and are scattered particularly over the extensor surface of the extremities more or less symmetrically, but also on the trunk and sometimes even on the face. They usually number from about ten to thirty, but I have counted over one hundred and fifty in one case (Fig. 92). The skin over them is of normal color, or pinkish, or brownish-purplish, raised or on the contrary slightly depressed and on pressure may present the appearance of orange-peel. Their consistence is very hard, or somewhat doughy; with rare exceptions they are painless. Very rarely, some may become eroded on their surface and covered with a crust. They are observed at all ages, in both sexes, and are not very uncommon [in France]. They may disappear spontaneously.

These disseminated sarcoids possess a great analogy with scrofulotuberculous gummas in the firm stage; they differ from them in that they do not soften and ulcerate; but these two pathological types may exist together.

They also possess such marked analogies with the erythema induratum of Bazin that I have thought these two affections might be identical; erythema induratum would then be merely a particular form, an objective appearance of disseminated sarcoid. It is, however, entitled to separate mention.

4. **Erythema Induratum of Bazin.**—Under the name of *érythème induré des scrofuleux*, Bazin had described more or less deeply indurated and imperfectly outlined, pinkish, red or purplish patches, which are observed in youthful individuals, especially young girls, almost invariably occupying the outer and lower portion of the legs; they may also be observed on the thighs, on the upper limbs, and even on the forehead. Their course is subacute, with congestive exacerbations accompanied by throbbing pains; periods of retrogression occur under the influence of rest.

Fig. 92.—Disseminated nodular sarcoids on the thighs and lower abdomen of a man aged forty-five years.

English authors, Colcott Fox and especially J. Hutchinson, have shown that Bazin's disease, as they call it, not infrequently gives rise to sluggish and obstinate ulcers (Fig. 102); this is the English type, or *Hutchinson's type*, which in this respect distinctly differs from the sarcoids and approximates the tuberculous infiltrations.

The *histological structure* of the various sarcoids referred to above is not uniform. The structure of the cutaneous sarcoid of Boeck, or lupoid, is entirely distinct. The hypodermic sarcoids of the Darier-Roussy type are to the highest degree tuberculoid in structure (Fig. 93), perfect tubercles [in the pathological sense] being found

in addition to the ordinary lesions due to reaction of the adipose tissue, known since Flemming under the name of "Wucheratrophie" (proliferative atrophy). The disseminated sarcoids have either an analogous structure, or that of inflammatory newformations; the whole gamut of lesions may be met with from erythema nodosum to the most characteristic tuberculous gummas. In erythema nodosum, the infiltration is also more or less ordinary, or else tuberculoid with partial necrosis. Thibierge and Ravaut, Mantegazze and Guibert emphasize the vascular lesions, which I was always able to demonstrate.

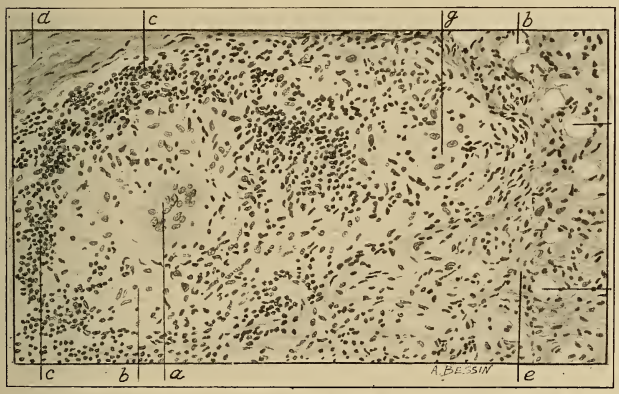


FIG. 93. — Histology of hypodermic sarcoids. Tuberculoid focus at the periphery of a lobulated nodosity of the thoracic region. *a*, giant cell; *b*, zone of epithelioid cells; *c*, *c*, zone of lymphocytes; *d*, fibrous tissues surrounding the nodosity; *e*, sclerosis of the adipose tissue; *g*, collection of epithelioid cells belonging to a neighboring tubercle. × 130.

The *nature* of the sarcoids is obviously infectious and the pathogenic mechanism of their origin is undoubtedly through embolism. But their etiological connection with Koch's bacillus, which would make them tuberculoses or tuberculides, has not been established, at least not for all types. Histology, as is now known, does not settle the question, for the tubercle is by no means characteristic of the bacillary infection, but may occur in the mycoses, in syphilis, leprosy, etc. Inversely, the absence of tubercles, according to numerous recent contributions in no way excludes the presence of Koch's bacillus.

As regards erythema induratum, its tuberculous nature may be said to be an established fact based on its common coincidence with visceral, bony, glandular, or cutaneous tuberculosis; or with papulo-

neurotic tuberculides and lichen scrofulosorum; and from its reaction to tuberculin. Still more direct proofs have been furnished; its tissue infects inoculated guinea-pigs with tuberculosis (Thibierge and Ravaut, Carle) and, in a few cases, the bacillus has been demonstrated therein (Philippson, Jadassohn).

Notwithstanding coincidences and transition forms, no such definite conclusions can be formulated in regard to the other types. It is still desirable, in cases coming under observation, to look for the Wassermann reaction, which is negative as a rule, and for the tuberculin reaction which is generally positive, as well as to carry out the necessary laboratory tests. The solution of this problem belongs to the future.

The *treatment* of sarcoids with arsenic has been vaunted as well-nigh specific; arsenobenzol is sometimes remarkably successful (Ravaut); I have obtained excellent results from calomel injections; at the same time as Thibierge, I became impressed with the rapid efficacy of tuberculin injections in very minute doses, especially in erythema induratum; as a rule, a combined medication with novarsenobenzol and tuberculin gives the best results. Rest and good general hygiene are imperative. In spite of treatment, recurrences are to be anticipated.

The term **paraffinoma** has been applied to hypodermic lesions which are often caused by injections of paraffin; in certain respects, these may be compared with artificial or induced sarcoids. About fifteen years ago vaselin injections and then injections of paraffin at a melting point of 65° or 55° were recommended for the correction of various congenital or acquired deformities, saddle-nose, misshapen ears, atrophic rhinitis with ozena, bad scars, fistulas, and even for the esthetic improvement of the face, the base of the neck or the female breast.

These injections are not devoid of danger. After a few years, a progressive swelling with a coppery redness and very painful tension may make its appearance at the injected points. Its cause is easily recognized from the site and the statements of the patient.

Histological examination shows a diffuse, nodular, chronic inflammation, with microscopical abscesses and giant cells, caused by the paraffin which has spread between the tissue-meshes and hollowed out cavities lined with endothelium. Injections of vaselin or camphorated oil sometimes produce analogous lesions. Massage, electrolysis and aspiration are usually inefficient and it is necessary to resort to surgical ablation.

CHAPTER XV.

ULCERATIONS, ULCERATIVE DERMATOSES AND CUTANEOUS GANGRENES.

Ulcerations of the skin are losses of substance due to a pathological process of molecular destruction or of gangrene, in contradistinction to wounds, which are directly produced by a trauma.

The term ulcer is reserved more particularly for chronic ulcerations with a marked tendency to persist a long time or indefinitely.

Cutaneous gangrene is the mortification or necrosis of a more or less extensive portion of the tissues.

I shall first discuss ulcerations in general and the ulcerative process; then the ulcerative dermatoses; finally, gangrene.

ULCERATION IN GENERAL.

Clinical Features.—The various ulcerations are differentiated by numerous features, which must be kept in mind for the diagnosis.

1. The variable depth of the loss of substance permits a distinction between: excoriations, which are superficial, involving only the epidermis and often resulting from a blister, a superficial pustule, or a vesicular process; they heal without a scar, leaving a simple pigmentary macule; and true or dermic ulcerations, which encroach upon or destroy the derma and are necessarily followed by scar-formation.

2. The extent of the ulcerations is rarely of much importance; it is useful to know, however, that certain varieties never acquire large dimensions, whereas others on the contrary may be of almost indefinite extent.

3. The configuration is geometrical or irregular, rounded, oval, polycyclic, reniform, circinate, etc.

4. The borders may be either sharp or imperfectly outlined, perpendicular, detached or overhanging, sloping, raised or flat, slanting, everted, fissured, etc.

5. The floor may be level or uneven, granular, papillomatous, "worm-eaten," sinuous, crateriform, raised, etc.

6. The color of the floor of ulcerations is red, purplish, grayish or yellowish.

7. The discharge is serous, purulent, hemorrhagic or sanious, and more or less profuse.

8. The crusts which result from the drying of the exudate present a variable appearance. Ostreaceous crusts, which are thicker in the center and apparently made up of heaped up disks of diminishing size, deserve special mention. They are characteristic of the lesion described as rupia by older writers (Fig. 94). Their occurrence is noted on ulcerations which increase intermittently, such as certain syphilides, more rarely in ecthyma.

9. The base of the ulcerations is sometimes soft, edematous, in other cases infiltrated and more or less indurated.

10. The periphery varies in color, consistence, condition of its surface, etc.

11. Tenderness, spontaneous or induced, or on the contrary anesthesia, are peculiar to certain forms.

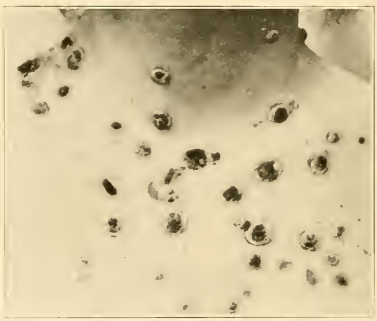


FIG. 94.—Ostreaceous crusts in a case of ulcerative secondary syphilides. Syphilitic rupia of old authors.

12. The condition of the corresponding *glands* deserves attention; so also (13) the site and (14) the course of the ulcerations.

Among these various features, those relating to the borders, the configuration, the base and the periphery possess the greatest value. They may afford information, to a certain degree at least, as to the process involved or show if the ulceration has occurred in healthy skin or at the expense of an infiltration or a tumor.

As to the topographical seat of the ulcerations, this is one of the signs most relied upon by the practitioner, often subconsciously, but not without reason. The fact that the lesions are situated, for example, on the face, the genitals, the legs, or the extremities, in itself supplies a valuable indication, often limiting the field of the diagnostic possibilities. The only feature of ulcerations, however, which in my opinion can be utilized for a rational classification, is represented by their course.

Pathogenesis.—The mode of formation of ulcerations is variable. Exceptionally, they result from mortification *en masse* of a more or less extensive portion of the tissues; this is what occurs in *the gangrenous dermatoses.*

Sometimes a dermic ulceration is derived from deep extension of an excoriation; or it may apparently develop in healthy skin; most commonly it is the result of disintegration or partial necrosis of an infiltration (tuberculous syphilitic, leprous, etc.), or of a tumor (epithelioma, mycosis fungoides, etc.).

In all these cases, the loss of substance results from the necrosis or necrobiosis of the cellular elements and of the tissue framework, followed by their molecular disintegration.

Without entering into unnecessary details, it is sufficient to emphasize that the conditions of the ulcerative process, as far as they are known, may be of two orders. One of these conditions concerns the germ or virus which may at the same time possess a toxic, necrotizing and digestive or lytic property and be able for a long time to resist the defensive measures of the organism; as is the case in soft chancre and very probably in phagedenism.

The other condition refers to the soil and consists of an imperfect blood supply, due to preëxisting regional vascular lesions (varicosities, etc.), or to the effect of the pathological process itself (tuberculosis, syphilis). Sometimes a general metabolic disturbance may be held responsible [diabetes, etc.].

Disturbances of innervation undoubtedly also play a part, not yet fully elucidated but proved by the so-called trophic ulcers, such as malum perforans, ulcers of nerve-leprosy, etc.

Different pathogenic factors are often combined.

Etiology.—In a general way it may be stated that all agents injurious to the tissues may, through their prolonged activity, give rise to and maintain ulcerations. They are divided into mechanical, physical, chemical and microbic agents.

Repeated traumatisms, by tools, shoes, bandages, or badly fitting apparatus, produce ulcerations. Their seat and form are often suggestive. A particular instance of these traumatic ulcers is furnished by the setons or issues, which our forefathers believed it advisable to establish by means of simple or epispastic agents.

As regards the induced [or artificial] ulcers which are observed on self-mutilators and malingerers and which were formerly referred to an assumed hysterical pemphigus, these are outside of any possible general description, in view of the variety of means employed and their results.

Crevices, rhagades, fissures or cracks are usually grouped among ulcerations, although not resulting from a loss of substance; they are rather linear wounds of traumatic origin in a dermic tissue

the elasticity of which may, however, have been impaired by a preceding inflammatory process.

They are met with on the extremities, on the hands and feet, as complications of a hyperkeratosis, or at the circumference of the natural orifices, at the lips, especially in scrofulous children, at the nipples of wet-nurses, very frequently at the anus, on the prepuce of diabetics, etc. Under the influence of movements of extension, the keratotic or macerated, sometimes even eczematized epidermis cracks and the lesion reaches down to the cutis. The borders of the crevices are perpendicular, the floor is bright red, sometimes bleeding; pain is often very severe; healing may take place without [visible] scars.

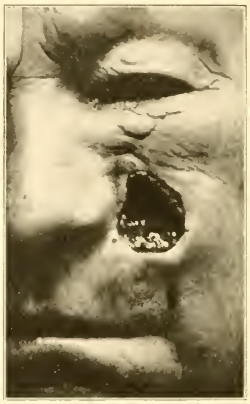


FIG. 95.—Tubular epithelioma in the form of ulcus terebrans.

The pain of cracks or crevices is relieved by local anesthetics, cocain and analogous remedies; superficial cauterizations with [a 5 per cent. solution of] silver nitrate may sometimes prove useful; it often suffices to apply either poultices or emollient dressings, or pastes or varnishes containing keratoplastic or keratolytic agents, according to the requirements of the case.

Among the ulcerations resulting from *physical* agents, those due to burns, frost-bites, *x*-rays, etc. (Chapter XXIII) may be mentioned.

Various toxic agents produce pustulo-ulcerative or primarily ulcerative lesions, either through ingestion, or more often as the result of external application. As an example may be mentioned the occupational arsenical ulcerations, occurring especially on the hands

and sometimes on the face and the genitals of laborers who handle arsenic or its compounds [such as Paris green].

The principal factors of ulcerations are microörganisms, which are concerned either exclusively and primarily, or in coöperation with other causes. This matter will be referred to again in connection with the ulcerative syndromes in particular.

As regards the ulcerations of tumors, their objective features and their course are too closely connected with those of the causative neoplasms to render a separate description advisable. It is necessary to state, however, that in some exceptional cases, the ulceration so markedly exceeds the neoplastic process that the latter may remain unrecognized. This rarely happens in the various sarcomas, in mycosis fungoides or in lobulated epithelioma, but is on the contrary very common in tubular epithelioma (Fig. 95), which was and still is described in some of its clinical forms under the name of *rodent ulcer* or *ulcus epitheliomatosum terebrans.*

Ulcerative Dermatoses.—Based especially upon their course, I distinguish between the following groups:

A. **Acute Ulcerative Dermatoses.**—They develop rapidly in healthy skin, never assume large dimensions, suppurate frankly and freely and are usually due to an external local infection. Soft chancre is typical of these dermatoses.

B. **Subacute Ulcerative Dermatoses.**—They are derived from one of the great subacute infections, more particularly syphilis, tuberculosis and leprosy. The ulceration takes place at the expense of a preliminary newformation.

C. **Phagedenic Ulcers.**—This name is applied to ulcers of rapid spread and great chronicity.

D. **Chronic Ulcerations, or True Ulcers.**—Their pathogenesis is apt to be complicated.

E. **Ulcerations of Mucous Membranes.**—The excoriations and ulcerations of mucous membranes are discussed in a separate paragraph, because it seemed to me convenient for the reader to find in one place important data on the diagnosis of lesions of the mouth and of the genital organs; these data will practically supplement what has already been stated on this subject in Chapter XI.

ACUTE ULCERATIONS.

Soft Chancre.—Soft chancre (ulcus molle) or *chancroid,* which will be described elsewhere from the nosographical viewpoint, develops very rapidly, and at the end of two or three days possesses its typical features.

It is a round or oval, relatively deep ulceration, the size of a pinhead, a lentil, a dime, or rarely larger. Its borders are perpendicular,

undermined or slightly detached; often they are cracked or fissured. The floor is of a creamy yellow or yellowish gray, irregular, "worm-eaten." The base is soft, sometimes doughy, but not parchment-like or cartilaginous. The circumference is red, slightly swollen.

Soft chancre has an abundant, distinctly purulent discharge. It is rather painful on touch or when the skin of the region is rubbed in moving. It is rarely single; as a rule, several are found, originating from the same contagion or from successive auto-inoculations. This was humorously expressed by Ricord when he said that the soft chancre is a family man and lives surrounded by his children. In some cases, in persons suffering from the itch, for instance, over a hundred soft chancres have been counted on the same subject.

The corresponding lymph glands are usually swollen and painful, one among them often greatly preponderating. This glandular swelling has a marked tendency to suppurate and discharge, undergoing transformation into an ulcer representing the so-called chancroid bubo of French writers.

Soft chancres are situated in the genital region in the vast majority of cases. They may occupy any point of this region; in men, the prepuce, the sulcus coronarius, the frenum, which is very often perforated, then divided, or the skin of the penis are involved; in women, the vestibule, the labia minora, the fourchette, the preputium clitoridis and frequently the anus. In the last-named localization, which may result from auto-inoculation, the chancre frequently appears double, the two folds of the ulcerated surface resting against one another, while its swollen lower border produces a protuberance resembling a condyloma.

The neighboring regions—intergluteal fold, internal aspect of the thighs, pubis, etc.—are affected in a considerable number of cases.

Extragenital Soft Chancres are rare. They may be observed on the fingers or on the hand (Fig. 96), where they are often misinterpreted; also on the face, although the existence of cephalic soft chancre was formerly denied.

The *diagnosis* is often easy, on account of the distinct objective features, the topography, the usual multiplicity, the rapid course of the ulcerations and the painful glandular swelling.

In doubtful cases, absolute certainty can be secured by means of two procedures: (1) Demonstration of the specific streptobacillus in the pus; (2) experimental auto-inoculation, which is valuable in that it provides a conclusive answer within forty-eight hours. This inoculation should be made in the deltoid region; as soon as it has been recognized as positive, the focus must promptly be destroyed by means of the thermocautery. In all doubtful cases, such as will be described, the employment of these two diagnostic

procedures, at least of inoculation, is imperative, as the treatment of soft chancre demands much more energetic measures than are necessary in the case of the ulcerations which may simulate it.

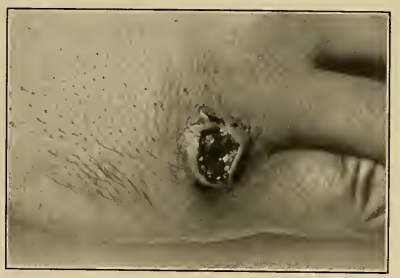


FIG. 96.—Soft chancre of the back of the hand.

Other Acute Ulcerations.—Mixed Chancre.—The mixed chancre of Rollett is not common, but has been observed with some frequency during the war. It results from inoculation at the same point with the bacillus of soft chancre and the spirochete of syphilis. When this inoculation has been simultaneous, a soft chancre develops first; at the end of two to four weeks, the incubation period of syphilis, the base of this chancre becomes distinctly indurated, its surface is modified to resemble that of a primary lesion and a generalized adenopathy develops. The demonstration of the spirochetes in such cases is very difficult and the diagnosis usually depends on the appearance of a positive Wassermann reaction.

When the inoculation is successive, namely when an indurated chancre becomes infected with virus of soft chancre, which is uncommon, the transformation occurs in the opposite direction. One must avoid the frequently committed error of applying the name mixed chancre to a soft chancre occurring in an already syphilitic subject, who therefore presents from the start a positive Wassermann reaction. The treatment must of course be both local and general.

Ulcerative Syphilitic Chancre.—The primary lesion of syphilis is, as a rule, simply erosive and seems to be an insignificant "sore."

In exceptional cases, in weakened or exhausted, alcoholic or diabetic subjects, in localizations predisposed to irritation and superadded infection, indurated chancre becomes ulcerative, deeply invading the derma and burrowing both superficially and deeply. A "chancre terebrans" may perforate the prepuce or even the urethra in men, one of the labia minora in women, etc. The ulcer is painful; its irritable, proliferating, granular floor resembles that of a soft

chancre. The induration of the base, however, is often very marked; the purulent discharge is less profuse than in soft chancre, auto-inoculation does not occur and generalized glandular swelling supervenes.

Ulcerative Secondary Syphilides.—Their course may be very rapid, or subacute. I shall have more to say concerning them later.

Ulcerated Herpes.—Although herpes is primarily vesicular and ordinarily gives rise only to very superficial erosions, these may become deep and suppurate under certain conditions of territory or of improper treatment.

The differential diagnosis from soft chancre, often rather difficult, rests in such cases on the polycyclic arrangement of the lesions, their simultaneous appearance, the persistence of non-infected herpetic elements in the vicinity and especially on the negative outcome of experimental auto-inoculation on the arm.

Ecthyma.—This consists of an at first bullous or pustular, secondarily ulcerative lesion. It affects especially the lower limbs, sometimes the trunk and has no predilection for the genital region. The ulcers have gently sloping, not perpendicular borders.

In all the cases mentioned, except that of mixed chancre, microscopical examination of the pus in smears shows the absence of the streptobacillus; inoculation on the arm produces a superficial impetigo-pustule, without involvement of the cutis and healing of its own accord in at most three or four days.

SUBACUTE ULCERATIONS.

Syphilis—As a rule, the secondary lesions undergo resolution, the tertiary lesions alone being destructive and having a tendency to ulcerate.

Sometimes, however, beginning with the first eruption, the papulo-crusted lesions ulcerate under the brownish or grayish crust which covers them; at the same time, more or less extensive and destructive ulcerations appear on the mucosæ. These cases are described as *syphilis maligna precox*. Weakened, undernourished or alcoholic individuals are predisposed to it; a special property of the germ has often been held responsible and tropical syphilis is of bad repute in this respect. However, this special course has also been noted in robust individuals who were infected by a syphilis of ordinary behavior. So much is certain that the spirochetes are usually rare in the malignant syphilides, although inoculation into monkeys may yield a positive result.

Ulcerative secondary syphilides constitute an irregularly scattered eruption of ulcerations which develop and enlarge rapidly; they have a round or more often oval form, perpendicular purplish

borders, a cupola-shaped floor, filled with sanguinolent pus, and a soft base. They are only slightly painful, except in certain localities.

Their peripheral extension and the dessication of the pus which they secrete, sometimes give rise to ostreaceous crusts, thicker in their center; this picture was formerly designated under the name of *syphilitic rupia* (Fig. 94).

FIG. 97.—Serpiginous tuberculo-gummous syphilides.

Discrete or profuse, sometimes even confluent in places, the secondary ulcerative syphilides sometimes co-exist with deep ulcerations of the mucous membranes and do not readily heal; they leave honeycombed dyschromic, sometimes mutilating cicatrices which constitute significant stigmata. They are usually associated with rapid emaciation, considerable cachexia, fever, albuminuria and pulmonary or digestive complications and may lead to death.

Malignant syphilis had been observed to be often rather resistant to mercury and iodide treatment, and in addition arsenic medication, injections of hypertonic sera, a strict hygiene and careful local dressings were recommended. The introduction of the arsenobenzols has improved the prognosis of this form of the disease; their efficacy is remarkable and rapid; it is recommended to combine their action with that of injections of soluble mercury or calomel.

Ulcerative tertiary syphilides are derived from tubercles, gumma, gummous infiltration, or sclerosing gumma. All of these, in contradistinction to the ulcerative manifestations of the secondary period, are generally localized or regional. Five principal types are distinguished:

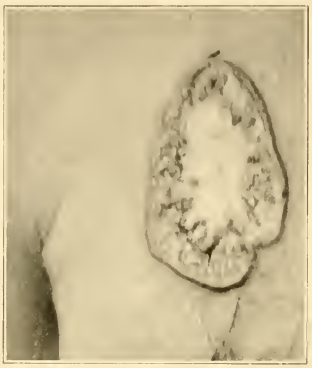


FIG. 98.—A typical syphilitic ulceration on the thorax near the right axilla.

1. *Tuberculo-ulcerative*, also known as *tuberculo-gummous syphilides* (Fig. 97) consist of rather large dermic tubercles, from the size of a pea to that of a hazel-nut, of firm consistence, but almost invariably terminating in ulcerative disintegration. Usually grouped in clusters, crescents, etc., these ulcerations develop excentrically and become arranged in serpiginous patches or areas of a highly characteristic appearance. These areas are honeycombed scars, checkered with whitish, purplish or earthy hues; the polycyclic border presents, sometimes only on one side, rows of deep ulcerations, round or in their turn polycyclic and punched-out, discharging a livid pus, or covered with thick, hard, adherent, sometimes ostreaceous crusts of a greenish-black color. The double polycyclic character of the patch and of its constituent ulcerations possesses marked diagnostic value.

2. *Syphilitic ulcerations* which may be called atypical have a less characteristic appearance. As a rule it cannot be determined whether or not they have begun under the form of tubercles; at

any rate, none are found at the circumference of the resulting lesion. This is usually a single ulceration, having the shape of an oval, a bean, an ear, etc., with regular margins describing fairly large curves, often with a moderately hard, raised border with an uneven rather shallow floor, sometimes cicatricial in the center (Fig. 98). Their distinct polycyclic contours, the indurated border, the parchment-like induration of their base, which, however, is not always easily demonstrable, the examination by biopsy, if necessary, serve to differentiate these tertiary ulcers from atypical tuberculous ulcers which will be discussed further on; they are much less deeply evacuated than gummous ulcers; their shape is more regular and their progress is decidedly more rapid than that of an epithelioma.

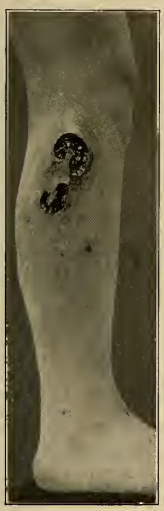


FIG. 99.—Syphilitic gummous ulceration.

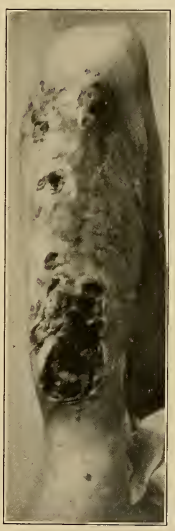


FIG. 100. — Syphilitic gummous infiltration, deeply ulcerated in places. The woman from whom this photograph is derived suffered from lesions of the same kind on the thighs, the thorax and the neck. Cure in two months through calomel injections.

3. *Syphilitic gummous ulceration* is a deeper lesion, with rounded or kidney-shaped contours, and loose, soft or edematous borders (Fig. 99). It results from the disintegration and evacuation of a hypodermic node.

4. *Ulcerated gummous infiltration* is of very variable extent, depth and configuration. It may extend to the tendons, the vessels, the periosteum, even the bones, causing very serious mutilations (Fig. 100).

5. *Sclero-gummous ulcerations* are those which originate in tertiary syphilitic, sclerotic or scar-tissue. They are observed especially on the tongue, though sometimes on the legs or in hypertrophic syphilomas. They result from a focal or a superficial necrosis, dry and caseous at first, later softening. Their abrupt onset, their irregular, sometimes angular form, the woody induration of their base, the slowness of their repair, are sufficiently characteristic.

These different types of *tertiary ulcerations* are connected by numerous intermediate types. There is always a more or less dense and circumscribed specific infiltration of variable depth which has undergone partial necrosis and purulent disintegration. Their clinical evolution is very irregular, sometimes sluggish, sometimes rapid. Their own characteristics commonly suffice for a positive diagnosis, even in the absence of a history or other evidence of syphilis, which, however, should always be looked for. The Wassermann test and sometimes examination by biposy may be required. In some cases the mycoses must be kept in mind. These tertiary syphilides are usually very amenable to the influence of specific treatment.

Tuberculosis.—The various types of ulcerations referable to tuberculosis differ in their appearance, their seat, their pathogenesis and also to a high degree in their virulence, but they are associated by connecting links.

Tuberculous Ulcer.—Typical tuberculous ulcers possess peculiar features which as a rule permit them to be quite easily recognized. Their seat of predilection is on the lips or tongue, or at any point of the mouth or pharynx; frequently also about the circumference of the anus; rarely elsewhere.

These ulcers are of ovoid, polycyclic or irregular shape, of an extent varying from a few millimeters to 1 or 2 cm., with wavy or ragged contours, perpendicular or detached borders, of a livid or purplish color, an uneven roughened granular floor, dotted with hemorrhagic points, often partly covered by grayish detritus. Tuberculous ulcers are almost invariably superficial, rather shallow.

On its floor or at its periphery may be seen the yellow granules of Trélat, punctiform or of pin-head size, in greater or less number; or gray miliary ulcerations.

The discharge is sanious and rarely profuse. The base is soft or slightly infiltrated. The regional glands are often enlarged. Always tender on pressure, tubercular ulcers may become distressingly painful when situated at points exposed to traction or friction. The

course is very slow, extending over weeks and months, without showing a tendency to spontaneous cicatrization.

The pathological anatomy of this ulcer is even more characteristic than its clinical appearance (Fig. 101).

In cases of rapidly progressive phthisis, tuberculous ulcers of the mouth may occasionally be seen to originate and extend within a few days, constituting *acute buccal phthisis*.

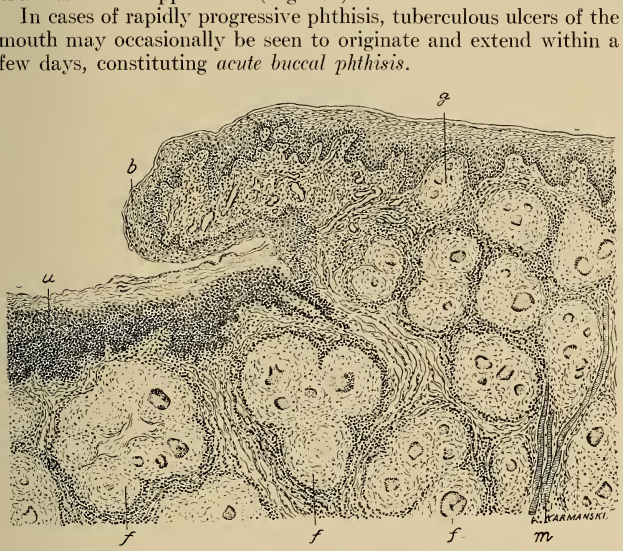


FIG. 101.—Histology of tuberculous ulcer of the tongue. Section from the margin of the ulcer. *b*, detached margin of the ulcer; *u*, floor of the ulcer, showing a light fibrinous layer which covers a tissue infiltrated with pus; *f*, *f*, conglomerated or isolated tubercles; they are usually composed of one or several central giant cells a collection of clear epithelioid cells and an external zone of lymphocytes and plasmocytes. None of these tubercles is in direct contact with the ulceration; *g*, a tubercle developed in a papilla; its enlargement or its confluence with other neighboring tubercles would give rise to a yellow granule of Trélat; *m*, striated muscle fibers of the tongue. × 40.

Sometimes, the ulcer assumes the characteristics of the *fissured variety* being elongated in the diretion of a fold and resembling a rhagade with one irregularly ulcerated portion. This is observed at the commissures of the lips, their median furrow, on the lateral borders or at the tip of the tongue, at points irritated by carious teeth; or again in the radiating folds of the anus, whence the ulceration may extend to the anal canal on the one hand and to the integument of a buttock on the other.

The *papillomatous ulcerative variety*, resulting from a combination of the ulcer with a process analogous to that of papillomatous or

verrucous tuberculosis, is likewise not uncommon. The bare or keratotic papillomatous elevations develop on the congested and cyanotic floor at the border of the ulcer. This variety is seen especially on the lips and at the anus.

The *diagnosis* of tuberculous ulcer is usually easy. *Traumatic ulcerations* heal rapidly when properly treated. *Soft chancre* is exceptional outside of the genito-anal region, has an abundant suppuration, a rapid course and is auto-inoculable. *Syphilitic ulcers* have a more regular form and an indurated base; in difficult or doubtful cases, recourse may be had to serodiagnosis and the demonstration of the bacillus. A suspicion of *ulcerated epithelioma* makes the microscopical examination by biopsy imperative.

Whenever the clinical features of the ulcer—although fairly characteristic—and the conditions under which it appears are not sufficient to remove all doubts, the diagnosis must always be confirmed by laboratory tests. The tissue-fragments obtained by scraping the floor, or better still from under the margins of the ulcer, contain bacilli; but it is necessary to scrape the tissues rather forcibly with a curette, bringing a little blood. Biopsy also must go rather deep. Inoculation into guinea-pigs may sometimes be necessary.

Lupus Ulcerations.—The ulceration of a lesion of lupus, whether delayed or appearing at once on a lupus exedens, always presents certain definite features which have been pointed out by Du Castel.

The borders are slanting, the adjoining tissue is purplish or brownish-yellow, tense, infiltrated, more or less transparent, soft, swollen, or covered with flabby proliferations.

The shape of the ulcer is round or oval, somewhat regular. Its floor is slightly depressed, of a lardaceous gray, light red or brownish color, sometimes proliferating; it bleeds easily, is of spongy consistence and is readily lacerated.

The base is the seat of a soft, non-plastic infiltration, more often movable than adherent to the subjacent tissues.

The secretion is turbid, sanious, drying in thin adherent grayish-yellow crusts, which are deeply imbedded and rarely ostreaceous.

Extension may be either superficial (*lupus serpiginosus*), or deep (*lupus terebrans*), or finally in all directions (*lupus vorax*).

Ulcerated Tuberculides.—Leaving out of consideration the minute punctiform ulcerations of the papulo-necrotic tuberculides, mention must be made of the sometimes extensive ulcers of *erythema induratum*. They constitute rather deep depressions, with variable borders, a grayish or mammillated and reddened floor and an extensively indurated base, all of which taken together with the purplish hue of the circumference and the seat of the lesion on the legs, invests them with a peculiar appearance (Fig. 102).

Occasionally they coincide with *scrofulo-tuberculous gummas*.

Atypical Tuberculous Ulcers.—Under this name I describe a clinical form which is not very uncommon and the diagnosis of which is always most baffling. These ulcers are usually met with in youthful individuals of the female sex who at the same time present either pulmonary lesions with a sluggish course, or cutaneous tuberculides.

FIG. 102.—Large ulcerated tuberculide of the thigh, in a young woman suffering besides from scrofulo-tuberculous gummas and papulo-necrotic tuberculides. Cure in six weeks, under the influence of rest, good hygiene, injections of tuberculin and aseptic dressings.

The ulcers are rounded, oval, or polycyclic, from the dimensions of a silver quarter to that of the palm of the hand, single or multiple, situated on any part of the integument. I have observed them on the shoulder, the neck, the chest, the groins, thighs, instep, etc. The irregularly outlined borders are often partly sloping, partly perpendicular or detached, of a dusky red or purplish color; the floor is grayish or bright red and proliferating; there are no yellow points, no lupus nodules; the base is slightly infiltrated and not indurated; they are absolutely painless. The course is extremely sluggish (Fig. 103).

The *diagnosis* lies between ulcerative tertiary syphilide, congenital syphilitic ulcer, abnormal tuberculous ulcer and ulcerated tuberculide. Laboratory investigations are required and yield

variable results. In the form of atypical tuberculous ulcer described by Marcel Lévy-Bruhl (Thèse de Paris, 1914), the histological lesions are clearly tubercular associated with vascular changes; the local tuberculin reaction and guinea-pig inoculation are positive.

In clinically analogous cases I was unable to produce tubercular infection in guinea-pigs. The degree of virulence of these atypical ulcers is accordingly not uniform. They are apparently due to bacillary emboli rather than to external inoculations. The treatment of election is surgical extirpation; aside from this, a very favorable action is exerted by tuberculin therapy and radiotherapy, combined with scarifications.

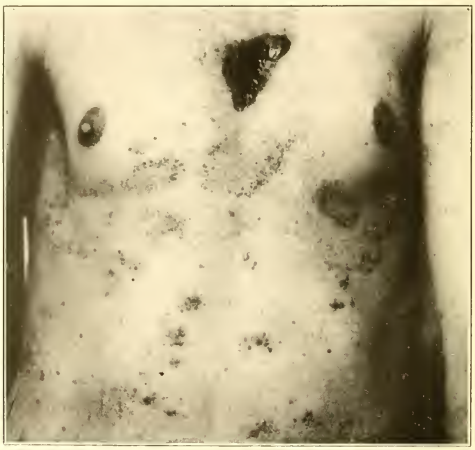


FIG. 103.—Atypical tuberculous ulcer of the presternal region, co-incident with lichen scrofulosorum in a girl aged seventeen years. Case of Lévy-Bruhl.

Leprosy.—The ulcers of leprosy have a variable appearance, course and pathogenesis. First, the ulcerated lepromas must be differentiated from tubercles or subcutaneous nodosities. They are observed especially in countries where leprosy is endemic, in severe cases, affecting neglected and badly cared-for individuals who drag along a miserable existence. The loss of substance results from partial disintegration of a bacillary infiltration, the remains of which are found at the circumference and at the base and becomes aggravated in consequence of vascular lesions and secondary infections. These lepromatous ulcers lead to serious mutilations.

Leprous individuals are, on the other hand, exposed to trophic ulcers, especially in the nervous or mixed types. Consecutive to, or associated with pemphigus leprosa, these are very superficial at first but obstinate and soon become covered with a rupioid crust; later on, they spread but retain a regular configuration, perpendicular walls and a membranous floor. They are hyperesthetic or anesthetic. When there are no other symptoms besides anesthesia, the ulcers are characteristic of Lazarine leprosy. Bacilli may be found in these ulcers at the period of onset.

Glanders.—The ulcerations which follow upon the abscesses or nodosities of chronic glanders, formerly called farcy, may present a fairly characteristic appearance, or on the contrary very closely simulate tuberculous, syphilitic or epitheliomatous ulcers

The features peculiar to them are: their irregular shape, their livid purplish margins, undermined and ragged as if torn by the teeth of mice, their very irregular sinuous floor, the softness of their base, their painlessness; furthermore, the presence of fluctuating nodosities or small abscesses at their periphery.

Mutilating glanders is apt to be situated in the center of the face, and causes considerable disfigurement and mutilation. The course is slow, irregular and progressive. The usual outcome is death.

The diagnosis can be surmised under certain conditions, but is positively established only by means of cultures and animal inoculation.

Dermatomycosis.—The ulcerations of sporotrichosis, blastomycosis, actinomycosis, and mycetoma (Chapter XXVIII) follow gummous nodosities, or sometimes after tuberculo-ulcerative and ecthymato-pustular dermic lesions. The pus contains the specific parasites, demonstrable on direct examination or by means of cultures.

Phagedena.—Phagedenic ulcers are characterized by a very marked tendency to invade the neighboring tissues, superficially or deeply, continuously or with interruptions; or rather at intervals; they are accordingly *acute* in their course and *chronic* in their duration.

These ulcers take their start in various lesions, as if due to the effect of a complication or a malignant change. The initial lesion is very often a soft chancre, rarely a hard chancre and not infrequently a tertiary ulcerative syphilide or a congenital syphilide; sometimes it is a lesion of ecthyma or a patch of spontaneous gangrene.

It goes without saying that not all extensive ulcerations must be grouped under the heading of phagedena—such as malignant secondary syphilides, tertiary syphilitic ulcers, tuberculous ulcers, cancerous ulcers, the ulcers of tropical diseases, etc.,—but only those which assume a both prolonged and rapidly destructive course,

not an ordinary attribute of their kind. The phagedenic ulcer of hot countries will be discussed further on.

Provided the necessary exclusions are carefully made, it will be seen that no matter what may be the original lesion, phagedenic ulcer presents a rather uniform picture, to be presently described. This observation has led the majority of writers to believe that phagedena is due to a superadded infection, a special bacterial agent, or a microbic association of particular virulence. Bacteriology having not as yet confirmed this theory, the conditions of the soil have been held responsible. It is true that phagedena sometimes attacks weakened or alcoholic subjects, but often also robust individuals; one is obliged to suspect in the latter a peculiar susceptibility or a deficient resistance against certain infectious agents. Although the majority of cases of phagedena occur in syphilitics, they do not all have syphilis. Age and sex are apparently irrelevant.

A phagedenic ulcer is of very variable extent, according to the activity and duration of the process. In a few weeks or months, starting from the genital organs, it may have destroyed the larger part of the penis or the vulva, reached the thighs, the perineum and the buttocks; have invaded a considerable portion of the abdomen and the back; in the face it may have destroyed the nose, the lips, a cheek, etc. It involves practically always the entire thickness of the integument; when it burrows deeply, exposing the muscles, tendons, large bloodvessels, etc., it is described as *terebrans;* it is called *serpiginous* when it becomes partly cicatrized while elsewhere progressing.

The configuration of the ulcer is sometimes irregular, often polycyclic. Brocq pointed out an objective appearance which is most characteristic and which he names *geometrical phagedena,* because its distinctly marked borders form perfect circles or ovals or parts of circles and ovals as if traced with a compass.

The borders, surrounded by a red and infiltrated zone, from 4 to 20 mm. wide, are perpendicular, sometimes undermined, with pockets of pus; in other cases sharply sloping. The floor, after it has been cleansed of the thick and yellow, rather than sanious pus which covers it, appears raw, red and granular, or sprinkled with shreds of sphacelated tissues. The center is sometimes epidermized and crusted.

The invasion is rapid, fulminating in some cases; as a rule, its progress is much more pronounced at some points than at others. The corresponding glands are usually swollen and painful, but they rarely suppurate. Although sometimes painless, the ulcer is more apt to be tender or even extremely painful on contact.

Histological examination shows a severe phlegmonous process

with a tendency to rapid necrosis, but a not very extensive range (Dominici, Rubens-Duval and Cl. Simon).

Bacteriology shows nothing but staphylococci, often streptococci, but no specific agent. Fuso-spirillary organisms and the Bacillus ramosus of Villon are absent. The search for anaërobes will have to be kept up.

Personally, in all cases which I have recently observed, the bacillus of Ducrey has been looked for without success; but I have always found the pus to be auto-inoculable on the patient's arm, producing a typical chancroid, only remarkable by a delayed development which may last from three to five days. [It is noteworthy that in these inoculated chancroids phagedenism does not develop]. This would lead me to assume that a large proportion of the most characteristic phagedenas, although evidently not all of them, are of chancroidal character. Caution is needed, for it is not in the nature of the chancre bacillus to remain indefinitely virulent in the same subject; furthermore, phagedena rarely gives rise to spontaneous auto-inoculations or chancrous bubos.

Although the appearance of phagedena, as previously stated, is nearly always uniform whatever its origin, it is desirable to point out some peculiarities of the various formerly admitted types.

Chancroidal phagedena is by far the most common. It originates in a soft chancre or in an open chancrous bubo and is therefore observed especially in the genital and inguinal regions. It does not possess the special features of chancrous ulcer, cracked or fissured borders, a yellowish worm-eaten floor, etc. I have seen it growing by true subdermal abscesses, opening at the surface or under the margins of the ulcer, where they form burrows and extensive purulent tracts. *Chancrous phagedena*, derived from a syphilitic chancre, is rare, unless one comprises incorrectly under this denomination the giant chancres and perforating chancres (terebrans) which may perforate the urethra in men, or the labia of the vulva, the lips, etc.

Tertiary phagedena, referable to a tuberculo-gummous syphilide or a gumma, has its seat of predilection in the middle of the face or on the genital organs. It may cause enormous mutilations, with fulminating rapidity, destroying, for example, the cheek, the nose, the upper jaw, the turbinates, the roof of the palate, hollowing out an enormous cavity in the middle of the face; the destruction is no less considerable in the pharynx or about the genitals. On the trunk and the extremities, it has been noted that phagedena often exposes, without attacking them, the large bloodvessels and the nerves.

The course of the phagedenic process varies in different cases, and even in the course of development of the same case. Partial cicatrization by itself alone is not a reassuring symptom, for extension may continue elsewhere or the cicatrix become eroded in

its turn. The total duration of the course of a phagedena usually extends over several years, up to four or five years, ten years or longer. The patients do not become cachectic, but the exquisite pain with each dressing causes them to become weakened and demoralized. Death from hemorrhage or septicemia rarely supervenes.

Treatment.—In the presence of genuine phagedena, in a syphilitic patient, general specific treatment must not be considered as sufficient. Mercurial medication, even when energetic and supported by adjuvants and excellent hygiene, usually fails; intravenous injections of arsenobenzol are positively indicated at the present day, but may prove insufficient.

In view of the rapidity with which phagedena exerts its destructive effects, local medication is imperative and must be very energetic from the start. The method of choice is the destruction of the infectious focus as a whole with superheated air, under general anesthesia. Extensive removal with the bistoury is likewise advocated, in the rare cases where it is practicable. When these measures are inapplicable, recourse must be had to cauterization with the thermocautery, zinc chloride, or tincture of iodin, which are of value only provided all the anfractuosities of the ulcer are reached. At the same time, or in the milder cases, repeated irrigation or washing with peroxide solution or hot potassium permanganate solutions. All kinds of antiseptic dressings and all varieties of powders have been tried. Brocq has obtained excellent results with a 10 per cent. collargol salve. [The Carrel-Dakin solution ought to give good results]. In these cases where the presence of chancroidal virus is probable or certain, no time should be lost with iodoform or silver nitrate, etc., but the heroic measures named above should be employed, notably hot air. Auto-inoculation on the arm, which experience has shown to be devoid of special danger, will serve to show if at a given point which may continue to appear suspicious after the general cauterization, virulent germs are still present and require renewed intervention. General tonic treatment and excellent hygiene, although insufficient by themselves, may favor the cicatrization after the ulcer has been transformed into a simple wound.

ULCERS.

Ulcus Cruris.—Ulcer of the leg, also known as *simple ulcer* or *varicose ulcer*, is a syndrome into the etiology of which a large number of conditions of various kinds enter.

The seat of predilection of simple ulcer is on the lower half of the legs, somewhat more frequently on the left leg, at the inner aspect and above the malleolus. It is usually single, sometimes multiple,

and in this case with a tendency to coalescence of the various ulcers; its shape is oval, polylobar or polycyclic and its extent may reach 15 and 20 cm. in diameter. Its floor is bright red or purplish, proliferating, oozing, or, in the absence of suitable care, covered with grayish detritus and sanious offensive pus. The borders are adherent, gently sloping or perpendicular, sometimes detached, thickened and callous (Fig. 104). The discharge is very variable, scanty and seropurulent when the patient remains in bed.

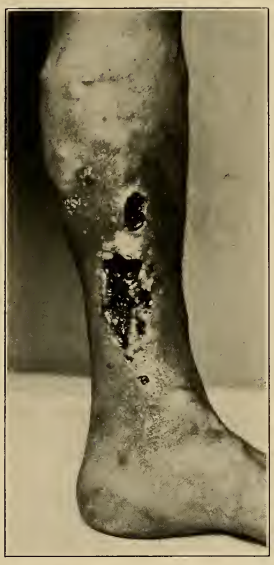


Fig. 104.—Varicose ulcer of the leg, with callous margins.

Sensibility is usually diminished and delayed on the ulcer and in its circumference, especially thermic sensibility, although painful and tactile perception are also lessened.

The periphery of the ulcer may be erythematous in a more or less extensive area, but otherwise normal; this is rare. As a rule, the neighboring and adjacent tissues are the seat of various changes, some depending on causes which have prepared the soil of the ulcer, others on secondary infections, unclean dressings, etc.

The complications consist of: Varicosities, which will be referred to again presently; varicose eczema, often circumscribed, which

may be dry, scaly, cracked, crusted, oozing, rubrum, impetiginous, etc., pigmentation of clay color, resulting from repeated intradermic hemorrhages; lymphangitis, more or less acute, or mild and recurrent; edema, sometimes soft and plastic, or more frequently non-depressible, elephantiastic—a hypertrophic callous state, or on the contrary a sclerotic atrophy. These last named lesions are entitled to a moment's attention.

Leg ulcer in the callous state has thickened, infiltrated, prominent borders, of cartilaginous or woody hardness; they slope rapidly in both directions and particularly steeply on the side of the loss of substance, which is often pale and covered with grayish detritus. The induration extends below the ulcer, and more or less far on the leg and foot; it involves not only the skin, but all the subjacent tissues, down to the bones.

These callous ulcers are sluggish, obstinate, extensive and not readily curable.

The fibrous induration may terminate in hypertrophy, in a secondary elephantiastic state, determined by inflammatory lesions of the lymphatics, bloodvessels and nerves. The bulk of the limb may become enormous and present deformities—which will be discussed under the heading of elephantiasis—with a supramalleolar cushion and swelling of the dorsum of the foot and the toes. The surface is smooth or hyperkeratotic, or more frequently papillomatous and verrucous.

In other cases the skin in the vicinity of the ulcer is in a state of diffuse or reticulated sclerotic atrophy; under these conditions it is thickened, smooth, tense, cannot be folded and presents a mottled, white, brown or purplish color. It is common for this dermatosclerosis to precede the formation of the ulcer.

The inguinal glands are often enlarged, the tendon reflexes of the limb may be absent; there is sometimes a slight muscular atrophy.

The nails of the foot are usually discolored, opaque, stratified, or even in a condition of pronounced *onychogryphosis*. It is not rare for the hairy system to be hypertrophied and for hyperidrosis to exist. These accessory lesions are often present on both sides, although the leg ulcer is unilateral in the majority of the cases.

Varicosities, which almost invariably accompany ulcer of the leg and exist on both lower limbs, rarely form large subcutaneous strands. The latter do not predispose to ulcer so much as the deep varicosities, which are not very visible and are not perceived unless the patient stands up, or by very careful palpation. They give rise, prior to the formation of the ulcer, to pigmentations in patches, reticulated, or in the form of an areola around a cicatrix; to telangiectasis; predispose to edema and eczema, and cause cramps, itching and a sensation of heaviness of the limb.

The ulcer begins in a variable manner; as the result of a wound, an ecchymosis, a small traumatic scar, a venous rupture, a small focus of phlebitis, an eczematous lesion, an impetigo or ecthyma pustule—all these lesions, instead of rapidly healing as they would on a well-nourished soil—persisting, becoming infected, deepening and enlarging under the crust which covers them.

It is exceptional for leg ulcer to begin as a plaque of spontaneous gangrene, although I have several times observed this occurrence.

Etiology.—Ulcer of the leg is observed especially between the ages of thirty-five and sixty years, slightly oftener in the male sex, in fatiguing occupations which require the erect position; in women who have had numerous pregnancies and in the poorer classes. The patients are, as a rule, atheromatous; sometimes polysclerotic, especially with renal sclerosis.

To the role played by deep varicosities as an etiological factor, established by Verneuil, has been added that of neuritis, deemed by Quénu to be itself of varicose origin; furthermore, that of arteritis.

It is readily understood that these general and local conditions impede the healing of the small above-enumerated lesions. This is not all, however. On this undernourished and poorly resistant soil, microbic infections are grafted and come to play the decisive part in the ulcerative process, in the recurrent lymphangitides, and in their sequel, dermatosclerosis.

Among the innumerable microörganisms of all kinds which flourish on these ulcers, the principal pathogenic agent seems to be most commonly the streptococcus. Leg ulcer has been described as "a chronic streptococcic chancre" (Sabouraud).

This local streptococcia is indefinitely prolonged, sometimes latent during long periods of time; not infrequently, the presence of the Bacillus pyocyaneus is likewise demonstrable.

Still other parasites when favored by special local conditions may undoubtedly give rise to a symptom-complex identical with that of simple ulcer.

It has long been noted that leg ulcers of typical appearance, in admittedly syphilitic individuals as well as in cases of undetected syphilis, will heal under mercurial medication. More recently, objectively similar cases have been recognized as *tuberculous*, through experimental inoculation. It is not known, however, to what extent these specific infections are involved.

Diagnosis.—The essential point consists, not in the recognition of the syndrome of leg ulcer, but in determining what part is referable to general and local predisposition as well as to the ordinary or specific infections.

Simple ecthyma is distinguished by the multiplicity of its lesions, their acute inflammatory character and their course.

Ulcerative syphilides and *syphilitic gummas* are usually multiple and bilateral, preferably situated on the external aspect of the legs. The former are often grouped in arcs of circles. Gummas begin as a nodosity and, after ulceration has occurred, are characterized by their rounded shape and undermined borders.

So-called *tuberculous ulcers* follow upon bony lesions or scrofulo-tuberculous gummas, or upon fungoid or lupoid tuberculosis, which are recognized by their special features.

Ulcerative erythema induratum of Hutchinson's type is met with only in youthful persons.

As stated above, these affections may lose their specific marks in a varicose and sclerotic territory.

Treatment.—This is medical or surgical. Innumerable methods and remedies have been advocated. In the first place, it is most essential to insist upon rest in the recumbent position, with the foot slightly elevated. The inflammation should be soothed and the ulcer cleansed by prolonged local baths, application of powders, aseptic or weakly antiseptic cytoplastic moist dressings. Strong antiseptics being rather injurious. [Liq. aluminii acetatis, one part to ten of water is one of the best of these moist dressings.]

After improvement has begun and the inflammation subsides, the formation of healthy granulations must be stimulated by means of dry dressings with inert or weakly stimulating powders, aristol, iodoform, iron subcarbonate, collargol, etc., the old styrax ointment, which has lost nothing of its value as a stimulant, as well as balsam of Peru.

Modern procedures may also be resorted to, such as light baths, radiotherapy, high-frequency currents, hot air; they sometimes exert a highly favorable action.

Imbricated strips of adhesive plaster, or better still, dressings with cotton or zinc glycerin-gelatin, which is valuable in these cases, or with elastic bands, may permit the patient's attending to some of his work. It is advisable not to change the dressings too frequently in leg ulcers that are healing.

The remarkably rapid healing of certain leg ulcers of ordinary appearance under the influence of mercurials, notably calomel injections, led to the adoption of the general rule always to give mercury a trial. At the present day, serodiagnosis should be called upon in all such cases and its indications be followed.

Surgical procedures, such as circumvallation, radiating incisions, etc., are merely mentioned by name in this connection, but it should be kept in mind that carefully and judiciously applied grafts may greatly shorten the time of cicatrization and furnish a better scar. In a few incurable cases of callous ulcer surrounding the entire leg, amputation has proved unavoidable.

After a leg ulcer has healed all efforts are in order to guard against recurrence. Roller bandages, elastic stockings, massage, local and general hygiene, may act efficiently in this connection. Electrotherapy in the form of ionization, combined with radiotherapy, is very serviceable for the control of dermatosclerosis.

Phagedenic Ulcers of Tropical Countries.—In the hot and moist climates of the tropical and subtropical zone of both hemispheres, ulcers of as yet undetermined character have been observed, which are called *tropical ulcer*, or according to the regions where they occur, *Annamite ulcers*, *Mozambique ulcer*, *malgache*, etc.

It is more than probable that this syndrome comprises altogether dissimilar types and has often been made to include ulcerative syphilides, chancrous phagedenas, leishmaniosis, varicose ulcers, etc. Tropical ulcer is usually situated in the lower third of the legs, or on the dorsum of the foot, very rarely on the hands, exceptionally elsewhere. It begins as a papulopustule which becomes gangrenous and spreads. It has rounded or oval contours, flat, unthickened borders and often a concave or funnel-shaped center.

An atonic form has been described; the sometimes very extensive loss of substance has a grayish, diphtheroid or sanious floor and a serpiginous outline; repair is delayed a long time and recurrences are common, even after several years.

The rapid, phagedenic or gangrenous form is extensive or perforating and advances in acute attacks, sometimes associated with fever; the ulcer is lined with soft fetid masses; it makes deep burrows, opens tendon sheaths and joints, exposes the bones, becomes complicated by lymphangitis or neuritis and gives rise to mutilations and faulty cicatrices. After a certain length of time, it may become atonic.

In both forms, a sensation of heat and sometimes intolerable pains are present. There is practically no tendency to spontaneous cure, except when the patient moves to a different climate.

Tropical ulcer is observed in wretched and exhausted individuals, weakened by alcohol, malaria, etc., especially in those who work with bare feet in water, as in rice plantations, etc.

The starting-point is an excoriation or a wound, the bite of a leech, or mosquito, a pustule of ecthyma, an ulcerative syphilide; briefly, any entrance for infectious microörganisms.

Tropical ulcer, although only slightly contagious, is inoculable and auto-inoculable; animals are not susceptible.

A variety of bacilli have been described. Le Dantec and others had observed a matting of bacilli; Vincent (1905) showed it to be caused by a fusospirillary association (*Spirocheta schaudinni*, Prowazek 1907), thereby proving its identity with hospital gangrene and ulcerative membranous stomatitis.

Treatment consists in first cleansing the ulcer by means of washing and suitable dressings, dry or moist, sometimes assisted by curettage. After it has been cleansed, washing with a zinc chloride solution, the application of calcium chloride as a powder or of protargol ointment of 5 to 20 per cent. (Castellani) or powdered arsenobenzol is in order, avoiding caustic agents and bland powders. Intravenous injections of arsenobenzol, although sometimes remarkably effective, may fail unless combined with good local treatment. In all cases, absolute rest is imperative and the general condition must be improved by appropriate measures.

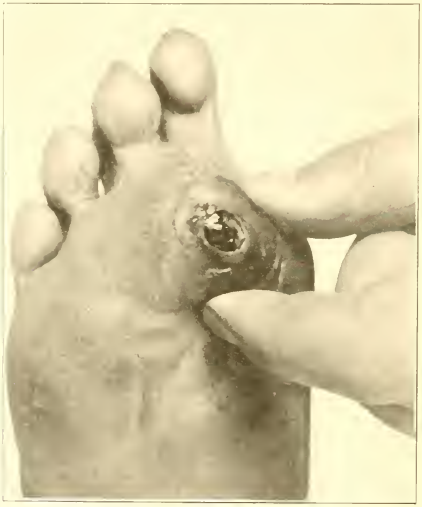


FIG. 105.—Malum perforans plantaris. Recurrence after a first attack which had necessitated amputation of the big toe and of the head of the first metatarsal.

From the prophylactic viewpoint, it is well to remember that in hot countries, all wounds including the most trifling abrasions should be carefully watched and dressed.

Malum Perforans. This name has been given to ulcers of small dimensions and very slow course, apparently connected with a trophic disturbance. They are observed in adults and aged individuals, oftener in the male sex, especially in the course of tabes, sometimes diabetes, syringomyelia, leprosy, polyneuritis, arteriosclerosis and sometimes without a demonstrable cause.

Malum perforans plantaris, studied by Nélaton, has its seat of predilection under the head of the first or fifth metatarsal, or at the heel, or on any other point of the foot exposed to pressure (Fig. 105). There are sometimes several ulcers and both feet may be affected.

The disease usually begins as a painful corn, a rounded hyperkeratosis on which blisters are repeatedly formed, or as a small crust. Before long, under the horny covering or under the crust, a rounded ulcer is found, with a proliferating or atonic floor, with perpendicular walls surrounded by hyperkeratosis. It burrows more or less deeply, and may reach the tendons, joints and bones and the latter when examined by *x*-rays are often found to be altered.

This lesion is painful on strong pressure, but often entirely anesthetic to needle-pricks in a variable radius.

Cases of mal. perforans affecting the fingers (malum perforans digitalis), the back of the foot, the nose or mouth, which may be encountered in the course of tabes, as well as mal. perforans of amputation-stumps, are very rare. They consist in losses of substance resembling traumatic abrasions, but occurring spontaneously, painless and anesthetic, often symmetrical, without any inflammatory reaction and indefinitely persistent. Buccal mal. perforans is often preceded or accompanied by a fall of teeth and frequently gives rise to losses of bony substance through absorption or sequestration.

The *treatment* of mal. perforans plantaris requires rest; the horny masses should be scraped off after moist dressings or a salicylic acid plaster has been applied, followed by curettage or cauterization of the ulcer. The wound is dressed with substances favoring cicatrization.

High frequency currents have seemed to me to be very efficient. In tabetic cases, healing is not infrequently obtained by intravenous injections of arsenobenzol. In order to guard against recurrences, which are always to be feared, it is advisable to protect the region against all pressure by means of appropriate dressings or special foot-gear.

In the grave cases associated with suppuration and opening of joints or bony necrosis, partial amputations are sometimes unavoidable.

ULCERATIONS OF MUCOUS MEMBRANES.

Although a comparison of the ulcerations of the various mucosa would be extremely interesting from the diagnostic point of view, the subject is too large for full presentation. I shall therefore discuss only the buccal mucosa (excluding the isthmus of the pharynx and consequently the anginas) and the genital mucosæ. On the other hand, the term "ulceration" will be used in its widest and most

comprehensive sense, so as to cover the most superficial epithelial abrasions up to the deepest ulcers. It must be conceded, however, that completeness is almost impossible in a subject of this kind.

Buccal Mucosa.—In considering an ulceration of the mouth, all its features must be carefully noted by the physician, but with special attention to the following points: depth, condition of the surface, configuration, base, number, site, course. Needless to say, the diagnosis will usually be greatly helped by the general examination of the patient.

1. **Dry Erosions or Depapillated Plaques of the Tongue.**—When these are persistent and not dependent upon a stomatitis, an acute infectious disease or a gastric disorder, they should suggest syphilitic smooth plaques, sclerotic glossitis, mild leukoplakia or glossitis marginata. There are still other smooth plaques the origin of which is not always demonstrable.

2. **Moist and Diphtheroid Erosions.**—All vesicles or bullæ of mucous membranes are very rapidly transformed through maceration and loss of the raised epidermic layer into erosions which are frequently covered with a pseudomembrane.

Buccal *herpes* is characterized by round microcyclic erosions, often grouped or confluent, rather painful, not very persistent. This herpes is recurrent in certain individuals.

Buccal *zona*, which is extremely rare, is said to be unilateral and non-recurrent.

Artificial bullæ, resulting from *bucus*, produced by a cigar, for example, are often of irregular shape and obvious localization.

Hydroa and *Duhring's disease* are accompanied, in about one-half of the cases, by buccal lesions on the inner surface of the cheeks, the posterior surface of the lips, the palate, sometimes on the tongue or on the isthmus of the throat; in the form of rounded or superficially confluent bright red erosions partly covered by a membranous or diphtheroid layer and extremely painful. The coexistence of the cutaneous eruption reveals their true character.

Chronic pemphigus has, on the contrary, a nearly constant and often primary buccal localization; it may remain the sole manifestation for many weeks.

In the few cases which have come under my observation, the pillars of the fauces, the pharynx, the cheeks and, later on, the lips and the borders of the tongue presented grayish or sanious, friable or adherent false membranes, covering partially ulcerative and oozing erosions, of irregular form and slowly progressive course. Objectively, the condition suggested diphtheria, Vincent's stomatitis, a medicinal stomatitis, or even syphilides. The pains were intolerable, the odor was offensive, the glands were enlarged; the general condition was seriously affected, with slight fever, but very

rapid emaciation. The diagnosis, rendered probable by these conditions and by the presence of a slight bullous elevation at the margin of the erosions, was confirmed by the appearance of blebs in the genital region and on the fingers. The gravity of this fearful disease is well known.

Syphilitic mucous patches are the most frequent buccal erosions and ulcerations and those which it is most important to know. The general description and that of the principal varieties are given elsewhere in this book (p. 620). It must be kept in mind that it is not justifiable to base the diagnosis of syphilis simply on the demonstration of lesions presenting the appearance of mucous patches, for they are not characteristic in themselves (A. Fournier); the coincidence of other manifestations with their special chronology is required or the demonstration of the Spirocheta pallida, a positive Wassermann reaction, generalized glandular enlargements, etc.

Syphilitic chancre is ordinarily erosive on the tongue. It is seen at the extreme tip of the organ, in the form of a fissured erosion; or on the dorsal aspect of the anterior third, in the form of a lenticular or oval, pink or sometimes grayish, single and painless eroded spot. Very careful palpation reveals a parchment-like induration at its base; the corresponding glands always assume a disproportionate enlargement as compared to the apparent importance of the lesion; they are non-inflammatory and painless; occasionally, the glands are the first to attract attention and point to the initial lesion. On the gums the chancre is likewise merely an erosion, frequently semicircular.

Buccal *diphtheria* is rarely primary, being generally secondary to diphtheritic angina. It consists of irregular pseudomembranous adherent patches, located on the lips and on the inner surface of the cheeks. The Loeffler bacillus is found, either in pure culture or in varied associations.

Diphtheroid stomatitis is of frequent occurrence in children; the false membrane here is less coherent; it is apparently often due to pyococci. The most distinctive form is impetigo buccalis (impetiginous stomatitis of Sevestre and Gaston), the features of which have been described elsewhere.

Thrush (soor, muguet) is not an ulcerative lesion, but might be confused with a diphtheroid erosion. It presents the appearance of a pure white or creamy adherent layer on an erythematous base; when it is detached, the mucosa is seen to bleed. Thrush is observed in cachectic individuals and in little children suffering from digestive disturbances. It occurs on the tongue, the cheeks, the isthmus, in the form of very small elevations which subsequently become confluent in ragged irregular patches. On inspection under the micro-

scope, the coating is shown to be made up of epithelial débris and very abundant filaments of endomyces albicans.

3. **Ulcerations.**—In *whooping-cough* (pertussis) of children, an ulceration of the frenum of the tongue is observed, caused by its rubbing against the teeth and assisting the diagnosis by its location.

Other *simple ulcers*, especially of traumatic origin, due to bites (as in epileptics) or to friction of the mucosa, are encountered at any age on the border of the tongue, or sometimes on the inner surface of the cheeks or the lips, notably at points which are in contact with some roughening of the teeth, due to dental caries, etc. These ulcerations are deep, of irregular shape with an edematous or sometimes indurated base, painful and usually without glandular involvement. They heal spontaneously after the removal of the offending tooth or roughness and disinfection of peridental foci of suppuration.

Common aphthæ are small, punctiform, or at most lenticular yellowish ulcerations, perfectly round or oval, following upon a grayish vesicle and surrounded by a characteristic carmine-red narrow border; they give rise to a very painful burning sensation. They are seen at all ages, always in very small numbers, but these aphthæ may come and go for a long time in dyspeptic and nervous individuals following errors in diet, overexertion, or irritative local causes in the mouth. The co-existence of herpes is not uncommon. Inflammatory complications are rare.

Common aphthæ are certainly in no way related to *aphthous fever*, a contagious disease of bovine origin, apparently transmissible to man through cows' milk [foot and mouth disease]. According to certain writers, this gives rise to an abundant eruption of aphthæ in the mouth, together with serious general manifestations; but this is not an established fact.

The *ulcero-membranous stomatitis* of Rilliet and Barthez and of Bergeron, which is now considered as identical with *Vincent's angina* and *stomatitis*, is due to mixed infection with the fusiform bacillus and spirilla; it is easy to demonstrate these parasites, which are seen in large numbers and often in pure cultures on smears of the exudate stained with dilute Ziehl's solution or carbolized thionin.

Vincent's stomatitis is observed particularly in children and youthful individuals, especially in weakened or exhausted persons, sometimes in almost epidemical form in crowded places, asylums, barracks, etc. [It was quite common among the soldiers in the Great War and was known as trench mouth.] The onset is followed immediately, or after a few days of fever, malaise, and local distress, by salivation, an offensive breath and pain.

Inspection of the mouth shows swollen and ulcerated gums; on the

cheeks, especially in the vicinity of the last lower molar tooth, then on the entire inner surface of the cheeks and sometimes of the lower lip, rounded ulcerations, or a sinuous ulcerated band, make their appearance. The lesions often invade the sides of the tongue, sometimes the palate and the velum or the tonsils; in some cases they remain unilateral or predominate on one side.

The following are some of their most essential features: the floor of the ulcers is grayish or the color of wine-dregs, usually masked by pultaceous, sanious, or even gangrenous detritus; perpendicular borders; a non-indurated base; the teeth are often bared through erosion of the alveolar margin; severe pain is present; mastication is impossible; there is profuse salivation and a peculiar disgusting odor.

The submaxillary glands are enlarged and painful; the expression of the face is drawn and pale; there is well-marked anorexia; a slight fever may be present.

When left untreated, the affection may last during several weeks or even months; by proper care, it can be cured in eight to fifteen days. The prognosis is on the whole favorable.

Syphilitic chancre of the mouth, in contradistinction to tonsillar chancre, is very rarely ulcerative.

Ulcerative *secondary syphilides* are observed in the ordinary forms of the disease as well as in precocious malignant syphilis and are characterized by their sharp outline circular form and by the depth of the lesion; they develop rapidly.

The *ulcerative gummas* of the tongue and the *sclero-gummous ulcerations* have been discussed elsewhere.

The *ulcerations* of *leukoplakia* deserve special mention. In the patches on the tongue, as well as on the cheeks and the lips, there are frequently encountered, not only more or less persistent fissures, but also very peculiar chronic ulcerations which do not seem to have attracted the attention of authors. I have repeatedly called attention to the characteristics of these *leukoplasic ulcers*.

Their shape is irregular, often angular; their floor, of a bright red color, smooth or finely mammillated, is frequently raised on a plane up to the level of the borders, from which it is separated by a deep furrow cut out as if with a knife and brought into view by unfolding it.

Besides being a source of annoyance, obstinate to treatment and frequently recurrent, these ulcers are furthermore alarming because they give rise to the dread of epithelioma; it is reassuring that their base is not more indurated than that of the neighboring sclerotic surfaces. In my opinion they are due to a local nutritional disturbance of the mucosa, referable to the underlying sclerosis and arteritis.

In order to cure them, recourse must be had to calomel injections, intravenous injections of arsenobenzol, or to local mercurial injections of weak concentration. Putting the teeth in good condition and proper buccal hygiene are indispensable. I regard it as inadvisable to irritate these lesions by caustic agents, but have often had good results from the employment of radium or *x*-rays.

Trophic ulcer, malum perforans of the mouth, has been observed, especially in tabetic patients, on the alveolar margin and the bony palate.

Tuberculous ulcer has already been described. I repeat that the tongue and the lips are among its seats of election.

I shall not dwell upon the buccal ulcerations of very variegated appearance which occasionally occur in the stomatitides of uremia and diabetes, in scurvy and other severe infectious diseases or conditions leading to cachexia.

Mercurial stomatitis is probably merely a special instance of Vincent's spirillary stomatitis. In its milder forms, it is limited to superficial irregular ulcerations, covered with a white mass, situated at the neck of the last molar tooth and its immediate neighborhood, at the neck of the lower incisors and sometimes on the borders of the tongue. They are accompanied by profuse salivation, a diffuse doughy exudate and a characteristic fetid odor. In the severe forms, however, deep gangrenous ulcerations have been noted, involving the cheeks, the gums, or even the bones and sometimes true gangrene of the tongue.

Noma, or buccal gangrene, was described in former times as rather frequent in children between two and four years of age, especially as a sequel of measles or typhoid fever, sometimes occurring in epidemic form.

Noma in children was said to begin as a livid swelling of the cheek; the mucous surface presents one or several blisters, covering livid then grayish sloughs; their fall leaves an ulceration which rapidly spreads and burrows, with ragged margins and a sanious, irregular, horribly offensive floor, sometimes perforating the cheek. The teeth fall out and the bones become necrotic. Death supervenes in four-fifths of the cases, in eight to fifteen days.

The condition in all probability represented an ulcero-membranous stomatitis, due like hospital gangrene to a fuso-spirillary infection. The very grave cases to which the above description is applicable have fortunately become extremely rare.

In the tropics, among half-famished and improvident natives, cases of extensive gangrene of the lips and cheeks are observed, suggestive of noma and perhaps referable to the same etiology, but more probably the result of phagedena of various kinds.

4. **Ulcerative Neoplasms.**—In the cases of tuberculo-ulcerative syphilide of the buccal cavity, or in the less common cases of ulcerated lupus of the buccal mucosa, it is readily seen that the ulcerations are not primary, but develop on a neoplastic patch having the features described above. They are generally multiple, rounded, perpendicular, with a relatively hard base in case of syphilis; irregular, less deep, with soft borders, in case of lupus.

Epithelioma of the buccal cavity is, in the great majority of the cases, of the lobulated types. I have, however, seen numerous instances of tubular [basal-celled] epithelioma.

In the great majority of cases, epithelioma is situated on the tongue or on the lips, sometimes on the cheeks, the pharynx, the palate, the gums, the floor of the mouth. It begins under the papillary form, or as a fissure, as I have pointed out in connection with leukoplakia, and also as a discoid elevation from the start. It becomes eroded very soon and then excavated by an ulceration.

The characteristics of epitheliomatous *ulcer* are as follows: Almost invariably irregular shape; very uneven, proliferating, sanious, readily bleeding floor, sometimes interspersed with small yellowish or gray masses; raised, swollen or overhanging borders in lobulated epithelioma, perpendicular borders in the tubular form; the base is always indurated, forming the tumor. Glandular enlargement occurs early in lobular epithelioma and determines an unfavorable prognosis; it is absent or very much delayed in the tubular variety.

Actinomycotic ulcer of the mucosa has the same characteristics as in the skin. Yellow granules can be extracted from it. The reader is referred to Chapters XXVIII and XXIX for the buccal ulcerations of the sporotrichoses, leishmaniosis, etc.

Other tumors, notably the sarcoma known as *epulis*, or even benign neoplasms, may become accidentally ulcerated.

Treatment of Buccal Ulcerations.—In a general way, much can be accomplished by scrupulous hygiene for the prevention of ulcerations of the buccal mucosa. Prophylactic treatment consists in proper care of the teeth and gums; the mechanical removal of tartar, cleansing and filling of carious cavities, extraction of stumps, cauterization of fungoid or soft gums and adjustment of prosthetic apparatus, should be left to the dentist. The results achieved must be maintained by regular brushing with a soapy powder or paste or any good dentifrice.

These measures are positively required in syphilitics, from the beginning of mercurial treatment, in patients recovering from severe infections, in persons predisposed to herpes, aphthæ, etc. In addition there should be abstinence from tobacco, alcoholic beverages and irritating food.

As to curative treatment, only brief suggestions can here be offered. Erosions, opaline mucous patches and aphthæ are benefited by superficial silver nitrate cauterizations every second or third day; irrigations with dilute peroxide water or potassium chlorate. Soothing mouth washes are sufficient in cases of acute irritation. Mechanical cleansing with a wisp of cotton wet with alkaline or borated solution will suffice in thrush.

The ulcerations of ulcero-membranous stomatitis are treated with mouth-baths of dilute Labarraque's solution, 1 to 20; cleansing with a small stick wrapped in cotton, followed by touching with silver nitrate; with powdered arsenobenzol, calcium chloride in substance, powdered methylene-blue or iodine tincture. Internally, sodium chlorate, grams 2 per diem, is recommended. Intravenous injections of arsenobenzol are efficient only when combined with proper local treatment. [It is the method of choice in Vincent's infection.] It is highly advantageous to apply to syphilitic ulcers, together with the indispensable general treatment, the various topical agents enumerated above. The treatments suitable for the other cases have been discussed elsewhere.

Genital Mucosæ.—The majority of the erosive or ulcerative affections occurring in the mouth may also be encountered on the genital mucosæ or semimucosæ of both sexes.

This is especially true of *herpes*, which is common in this location; of *syphilitic manifestations* of all kinds and of *hydroa;* on the other hand, aphthæ, impetigo, thrush and diphtheria are rare. Again, localizations of various dermatoses are here met with, which do not occur in the mouth, such as eczema, psoriasis, eczematides, scabies and, furthermore, a few special erosions and ulcerations.

Traumatic excoriations due to coitus, scratching with the fingernails, rape, etc., often have a characteristic irregular or fissured form; unless complicated by a superadded affection they heal with remarkable rapidity.

Soft chancre has been sufficiently emphasized and does not require further discussion.

Gonorrhea gives rise, in men, to a more or less acute diffuse balanoposthitis, sometimes with phimosis and ordinary or follicular erosions at the base of the glans and in the sulcus. Gangrene of the prepuce in these cases is very rare.

In women, gonococcal vulvitis may be accompanied by small para-urethral follicular ulcerations, by *canaliculitis*, namely inflammation, sometimes ulcerative, of the excretory duct of Bartholin's gland, sufficiently characterized by its seat; finally, by *gonococcal erosions* of a dusky red, granular surface, distinct or jagged margins, localized especially outside of the carunculæ myrtiformes. Being frequent and very persistent, these lesions may at the first glance

betray the existence of gonorrhea, which the patient is endeavoring to conceal.

Diabetes gives rise not only to erythematous, eczematous, etc., balanoposthitis and vulvitis, but also to fissures and ulcerations of very variable aspect, syphiloid in appearance, and even to gangrenes.

Circinate erosive balanoposthitis manifests itself in the form of extensive polycyclic, superficially erosive lesions bordered by a narrow white margin. This affection, attributed to spirilla by Berdal and Bataille, was recognized by Queyrat as a localization of the fuso-spirillary symbiosis which also causes Vincent's stomatitis. An analogous affection may be met with in women. In neglected cases, circinate balanitis lasts several weeks and is often recurrent. It is very easily cured by cleanliness and painting with a 1 to 30 solution of silver nitrate.

Syphilitic chancre, the different varieties of *secondary syphilides* or *mucous patches,* and the *tertiary syphilides* of the genitals have served for the descriptions of the manifestations which I have already given.

Tuberculosis may manifest itself in the same regions, although this is very rare, as *tuberculous ulcer,* resulting from a contagion or auto-inoculation and as *ulcerated lupus,* which is still more rare.

Under the name of *esthiomène,* introduced by Huguier in 1848, a rare syndrome is still occasionally described, which is characterized by a chronic ulceration of the vulva associated with elephantiasis. The more or less deep and often painless ulcer is situated at any point of the vulva, preferably at the entrance of the vagina. The sometimes enormous hypertrophy involves the labia minora, the clitoris, the labia majora, the perineal region, etc.

It is more than likely that the nature of the ulcer is very different in different cases, so that there may be tuberculous or lupus ulcers (Bernutz, Ficquet), soft chancres of low virulence (Jacobi), ulcerative tertiary syphilides, analogous with anorectal syphiloma, or even epitheliomas, which as a result of local conditions, notably a lack of cleanliness, become infected and give rise to sclerogenic inflammatory edema. [An ulcerated infected lymphangioma of the vulva may produce a similar picture.] It has recently been pointed out that the clinical picture of esthiomene is perfectly reproduced in the *ulcerative granuloma of the genital organs.*

Ordinary secondarily infected wounds or excoriations may finally suffice for the production of this pathological type, as a sequel of lymphangitis, sclerosis, or removal of the inguinal glands, etc. Esthiomene would in this way become entirely comparable to ulcer of the leg with elephantiasis nostras.

Treatment of Genital Ulcerations.—No matter what may be the cause and character of a balanoposthitis, phimosis or vulvitis, it is of great importance to keep the parts as clean as possible. If the glans cannot be uncovered for the irrigations, on account of the existing pain and inflammation, injections must be introduced into the subpreputial sack by means of a flexible rubber nozzle, beginning with lukewarm cleansing injections with large amounts of water, followed by astringent injections with a 1 : 100 silver nitrate solution, for example, or dilute peroxide water.

Exposed erosions or ulcerations are treated by touching them with silver nitrate, tincture of iodine, etc., with bland non-fermentable mineral or medicated powders or finally with salves or creams.

Keeping in mind, however, that cauterizations and numerous topical agents produce an induration which may become misleading, these should not be employed unless the diagnosis is already quite positive.

In women, it is necessary to emphasize the manner in which the irrigations are to be used so as to be really efficient. It is often advantageous to prevent mutual contact between the parts, by inserting gauze or cotton tampons freely dusted with powders or covered with a layer of zinc ointment.

In case of important or serious genital ulcerations, notably those of gangrenous appearance, the surfaces must be cleansed by local baths, moist dressings, washing with peroxide water or camphorated alcohol; next, iodoform powder may be applied, with a little salicylic acid or camphor, or according to the case, calcium chloride, balsam of Peru, etc.

Needless to say, all necessary directions must be given for the avoidance of contagion.

CUTANEOUS GANGRENE.

Cutaneous gangrene means mortification or death of a portion of the integument and may be accompanied by the same changes in the underlying tissues. When it is abrupt and complete from the onset, it is named *necrosis;* when it results from a progressive loss of tissue vitality, it is called *necrobiosis;* this distinction is of small importance, the two processes being frequently combined. The "dead" portion is known as a *slough* or *sphacelus.* It is distinguished as *dry gangrene* and *moist gangrene,* according to its being dry and mummified, or on the contrary, moist, putrefying and fetid.

Gangrene manifests itself as a change in color of the skin, which at the same time becomes cold and anesthetic to touch, pricking, and temperature. In the case of dry gangrene, the skin is yellow or purple, then brownish, and promptly hardens through desicca-

tion, becoming depressed below the normal level. Moist gangrene often begins as a purulent bulla, or rather as a bulla with sanious or hemorrhagic contents, the floor of which becomes necrotic; or a flabby, mottled, grayish surface may on the next day become covered with sanious blebs.

While sometimes unnoticed by the patient, the process in other cases is preceded or accompanied by stiffness, tingling, heat and intolerable laminating or tearing pains.

After a few days, unless the necrosis proceeds very rapidly, the slough becomes surrounded by a congestive halo, sometimes bullous at its periphery; on the line of demarcation between the dead and the living tissue, a deep groove is formed which suppurates more or less freely. Having become dark brown or black, the sphacelus retracts and is finally cast off, exposing an ulceration covered with detritus, or sometimes pink and proliferating.

The *pathogenic mechanisms* to which the various forms of cutaneous gangrene are due, are multiple. In each particular case, there is reason to look for one or several of the following conditions, which, moreover, instead of excluding one another are very frequently associated:

A. Direct local action of a mechanical, physical or chemical necrotizing agent.

B. Suppression of the blood supply (through embolism, arteritis, etc.).

C. Serious alteration of the blood itself.

D. Necrotizing infection.

E. Serious change of the trophic nerve supply.

The last-named factor, often emphasized in the past (Zambaeo, 1859), is at present considered as altogether accessory. Neurotrophism may sometimes perhaps act as a favoring factor, but the existence of trophic gangrene or necrosis of trophic origin cannot be admitted. So-called hysterical gangrene is always artificially induced.

The pathogenic mechanism from which certain secondary gangrenes are derived, for example those which sometimes complicate erysipelas, anthrax, soft chancre, etc., or which originate in malignant tumors, cancers, sarcomas, the tumors of mycosis fungoides, etc., is probably complex and cannot always be definitely established.

A. Direct Local Gangrene.—This results from an injury directly affecting the mortified point. *Traumatic gangrene*, as a sequel of crushing, severe contusions, war-wounds, belongs to the domain of surgery. *Prolonged compression*, for example by a very tight plaster apparatus, may produce a direct slough or ulceration.

Decubitus may have the same effect on the compressed points, especially the sacral region and the trochanteric regions, much more

rarely the heels or the scapular regions, in patients whose nutrition is seriously impaired and whose vascular tension is lowered by a severe general disease or a disease of the nervous system (dementia, myelitis, hemiplegia, etc.).

The sloughs of decubitus [bed-sores], which were formerly considered as of trophic origin, are really due to the chronic irritation of the integument by contact with the urine, fecal matter, and cutaneous secretions, in very weakened or hypersensitive individuals, with secondary infection of the macerated and excoriated skin. They can generally be avoided by extreme cleanliness, aseptic powders, and the employment of air-cushions.

Physical causes, such as burns, frost-bite, contact with continuous current electrodes, high frequency sparks, *x*-rays in excessive doses, will produce gangrenous patches at the damaged points.

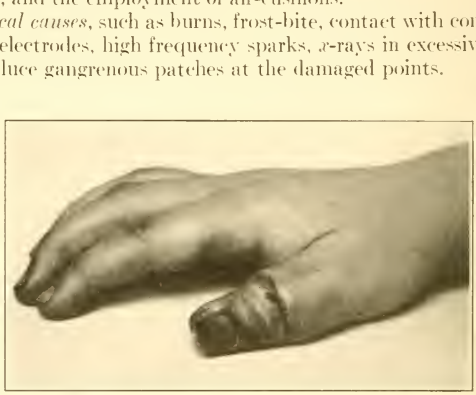


Fig. 106.—Carbolic gangrene of the thumb, in a child aged five years, who had worn a carbolic acid dressing during one night, for a scratch made by a cat.

Numerous chemical agents, so-called "caustics," notably strong alkalis and acids, certain salts, corrosive sublimate, zinc chloride, etc., will do the same. Sometimes, iodine tincture and even mustard plasters may produce sloughs, especially in children.

Carbolic gangrene is entitled to special mention, on account of its relative frequency and its insidious behavior. It is noteworthy that the total loss of a finger has repeatedly followed upon a simple panaris or whitlow, dressed with a solution of carbolic acid, even in supposedly harmless dilutions (Fig. 106). The patient is not warned by any painful sensation of the impending gangrene. It is therefore advisable to discard carbolic acid dressings, especially for the extremities.

In the direct gangrenes, the injurious factor usually acts at the same time on the tissue constituents themselves and by producing

stasis or thrombosis in the capillary bloodvessels. Infection, when present, is superadded.

B. Gangrene of Vascular Origin.—Any portion of the organism in which the circulation of the blood is completely and permanently arrested, inevitably undergoes necrobiosis or necrosis. The venous system, which is rich in anastomoses, is rarely affected; gangrenes in the course of phlebitis, or of phlegmasia alba dolens, being of exceptional occurrence. Almost invariably, the obliteration is arterial, affects the extremities, and the gangrene is of the dry type. Vascular rupture, sometimes ligature, compression, embolism, especially thrombosis through acute or chronic arteritis, produce different forms of progressive gangrene of the extremities.

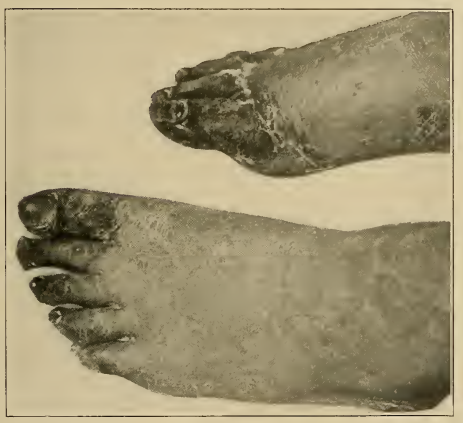


Fig. 107.—Dry symmetrical gangrene of the toes.

These gangrenes are observed especially on the feet, beginning with one or several toes, attacking at once the skin and the deep tissues, including the bones; hence they do not belong to the domain of dermatology. Only the principal types are here mentioned: Senile gangrene; gangrene due to obliterative endarteritis of Friedländer (Fig. 107); gangrene due to syphilitic arteritis [to endophlebitis migrans, Buerger]; gangrene in typhoid fever, etc.

Gangrene of the extremities, as a rule, partial or limited to the fingers or toes, which occurs in the grave forms of Raynaud's disease, is preceded by attacks of "local asphyxia" and "local syncope"; it has been attributed to a vascular spasm, a mechanism which would

probably be incapable of producing this result. Its pathogenesis is not known. The rare cases of gangrene observed in progressive scleroderma may be compared with it.

C. Gangrenes Through Changes of the Blood.—Under this heading I group the toxic gangrenes and those gangrenes which may be considered as autotoxic.

Ergot of rye gave rise in the middle ages to epidemics in which the general phenomena of *ergotism* were associated with tingling sensations or severe pains and mutilating gangrenes of the extremities.

Carbon monoxide poisoning is capable of producing gangrene in large patches, or involving an entire extremity. Chloral, when administered for a long time and in large doses, has been accused of the same effects. The question of gangrenes in the course of Bright's disease, which are rather exceptional, is not well understood.

Diabetic gangrene, on the contrary, is not uncommon. It is observed in individuals who are by no means cachectic, even in the enjoyment of excellent health and without large amounts of sugar in the urine. Assuredly neither hyperglycemia nor neuritis nor even arteritis can be held responsible, except perhaps in the massive forms. A slight traumatism or an accidental cutaneous affection often serves as determining cause. The principal part is probably referable to a local infection, streptococcic or other, or to septic embolisms.

Massive diabetic gangrene may abruptly attack an extremity, an entire limb, the genital organs, etc. In other cases, gangrenous *patches* are scattered over the feet, the lower limbs, or elsewhere, in the form of grayish or brown sloughs, preceded by a blister and progressively extensive. In this form—named *bullo-serpiginous*, by Kaposi—the center may heal while a bullous elevation at the periphery marks the zone of invasion. The course is rapid or on the contrary very sluggish. The prognosis in a general way is not very unfavorable.

When gangrene in diabetics is *secondary* to suppurative lesions, furuncles, carbuncles, abscess, etc., the symptoms of reaction may be mild, although the situation is really threatening.

D. Gangrenous Infections.—Infection through bacteria of various kinds, ordinary or special, aërobes or perhaps especially anaërobes, plays a considerable part in the origin and in the course of cutaneous gangrenes.

It seems logical, *a priori*, to group under two separate headings: (1) Secondary infected gangrenes, in which a lesion or affection of the skin, of definite character (wounds, ecthyma, furuncle, erysipelas, pemphigus, varicella, zona, syphilide, soft chancre, etc.), becomes gangrenous through the effect of an excessive virulence of its causa-

tive agent, a diminished resistance of the organism, or the introduction of necrotizing germs from the outside; (2) primary infectious gangrenes, resulting from microbic embolism in the skin by the hematogenous route. Sometimes, the direct examination of the patient or the history will show at first sight that the case belongs to one or the other of these groups; in other instances, there remains a doubt.

There is little advantage in dwelling upon the secondary or accidental infectious gangrenes. I shall therefore discuss only those which are primary and essential; leaving aside, of course, the gangrenes of the classified general diseases, such as anthrax, bubonic plague, etc., and discussing instead the clinical forms of dermatological character.

Multiple Gangrene of Children.—This is a somewhat unusual clinical type, comprising positively dissimilar forms, such as those cases which have been described under the names of *multiple cachectic gangrene of the skin* (O. Simon and Eichhof), *gangrenous dermatitis of children, gangrenous varicella, gangrenous ecthyma, gangrenous urticaria, purpura fulminans*, etc.

Instances have been observed in very young children, especially girls, and in weakened individuals. Sometimes, the impression of a special disease is conveyed; in other cases, one is apparently confronted with an extraordinarily malignant form of an exanthem (varicella, vaccinia, measles) or of a pseudo-exanthema (purpura, polymorphous erythema).

In a few days, a more or less abundant eruption of erythematous, urticarial or purpuric spots makes its appearance, or there may be bullæ containing a reddish serous fluid, or pustules, which enlarge and multiply. Their center very promptly turns black; the slough, concealed or not by a crust, spreads more or less, then becomes surrounded by a suppurative groove and loosens, leaving a perpendicular or dome-shaped ulceration with a sanious floor. The coalescence of several lesions gives rise to festooned patches. The sloughing may destroy a portion of the nose, of the lips, of the lobe of the ear, of the external genitals, or of the fingers and toes. Nodosities, edemas and abscesses may also be met with.

The eruption is situated especially on the lower portion of the trunk and on the thighs; it has another site of election on the neck, on the scalp, and on the face; but it may be widely disseminated.

The general symptoms are of very variable severity, sometimes only slightly marked; but often the condition is associated with a high fever, digestive disturbances, prostration, convulsions, as well as visceral complications leading to death; the mortality is about 50 per cent. In favorable cases, recovery is rapid.

Numerous pyococci and bacilli have been held responsible. In

the carefully studied case of Veillon and J. Hallé, the pathogenic agent was the *bacillus ramosus;* it is probable that other anaërobic microbes may produce analogous phenomena. Recently a few cases could be referred to the meningococcus.

Multiple Gangrene of Adults.—No discussion is here called for of the gangrenes with multiple foci, accompanied by abscesses and gangrenous phlegmons, which occur in rare instances in the course of pestilential diseases, very advanced cachexias and severe diseases of the nervous system. These are cases of septic embolisms with anaërobic microbes, of genuine metastases, derived from a bed-sore or a gangrenous focus of any kind, especially a pulmonary gangrene.

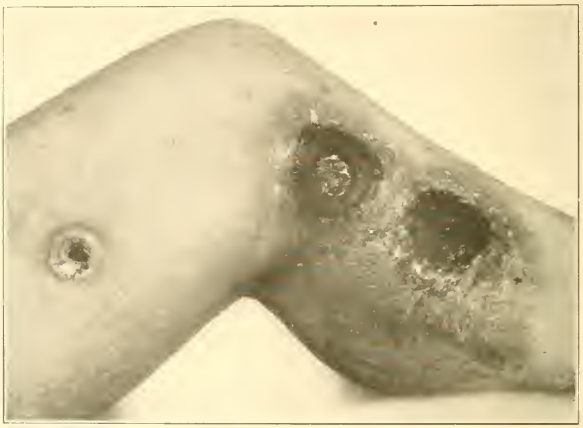


Fig. 108.—Multiple gangrene of adults; of fifteen days' standing, in a man aged seventy-one years in good health.

However, gangrenous eruptions entirely analogous to those of children, are also encountered in youthful individuals, in good health or slightly weakened and even in adults of middle age. They have the same onset, the same local symptomatology, the same general symptoms and develop in successive attacks. Examples have been quoted by Doutrelepont, Hallopeau, Carle, Brocq. I have personally observed several rather dissimilar cases, some with multiple patches limited to a single region (Fig. 108), others with very numerous lesions scattered over the entire integument. The prognosis is grave, although not necessarily fatal. Mention has been made of possible contagiousness (Demme).

Fulminating Gangrene of the Genital Organs.—A. Fournier has pointed out a rare type of gangrene, observed especially in young adults, leading to grave mutilations, sometimes to death.

Usually as the result of a slight excoriation, there suddenly develops, without paraphimosis, an enormous rose-colored edema of the penis and scrotum, with chills, high fever, etc. At the end of twenty-four to thirty-six hours, the penis after having assumed the appearance of a large "clapper," presents purplish, black or white gangrenous patches, which may destroy the entire sheathes, often the scrotum and rarely the corpora cavernosa; the swelling subsides at the end of about a week.

A very virulent streptococcus has been found in the serous discharge. These are probably cases of *gangrenous erysipelas*.

Treatment.—All cutaneous gangrenes are difficult to treat. Hygiene and general medications must not be too much relied on; even in the cases of diabetic gangrene, where a strict diet is imperative, this cannot prove sufficient by itself alone.

Locally, it is essential to supervise scrupulous cleanliness and well-applied protective dressings. In some cases, moist aseptic dressings are useful, or the application of dilute peroxide water, or even prolonged local baths; but as a rule, dry or oily dressings are preferable. Dressings with strong antiseptics are frequently injurious.

The parts may be swabbed with camphorated alcohol, or with a weak solution of potassium permanganate, followed by wrapping in dry sterilized cotton; or large quantities of aseptic or weakly antiseptic powders may be used (quinine, iron subcarbonate, dermatol, iodoform). Oily dressings, with carron-oil, guaiacolized oil, etc., are often better liked and seem to act more favorably.

When the gangrene is progressive it may be of vital importance to destroy the focus of putrefaction and the neighboring zone of invasion. This is accomplished by applications of superheated air in jets under pressure; this requires special apparatus, experience and skill, but actually constitutes the best treatment against extensive gangrenes, diabetic as well as others. The hot-air method is preferable to surgical removal, which will only be called for after the process has been checked, for the resection of sequestra or the trimming of stumps.

CHAPTER XVI.

DYSCHROMIAS.

Cutaneous Pigments.—The human skin is normally pigmented throughout; in this respect, there are merely differences in degree between the white race and the colored races, between the different regions of the body and between individuals of the same race.

The physiological cutaneous pigment consists of extremely small brown or black granules of an organic substance named *melanin,* which contains no iron, but sulphur in variable proportion. The melanin granules are situated in the basal layer of the epidermis and, furthermore, when the pigmentation is very marked, in certain cells of the papillary body; the color of the hairs and of the choroid is likewise due to melanin.

Under pathological conditions, two other pigments may be met with, *hemosiderin,* an ochre pigment which gives the reactions for iron and is formed in bloody extravasates, becoming deposited exclusively in the cutis; and the *paludean* pigment which is characteristic of malarial melanodermia.

Except in cases which will receive special consideration, the dyschromias are due to variations in the amount of melanin.

Dyschromias.—The name "dyschromia" is applied to pathological changes in the color of the skin, resulting either from an excess or a deficiency of pigment. They do not disappear on pressure with the finger, resist all washing, etc., and ordinarily persist a long time, sometimes throughout life.

As a rule, there is an excess of pigmentation. These *hyperchromias* may be circumscribed, representing pigmentary spots; or diffuse, representing melanodermas.

Hypochromia and *achromia* are more uncommon.

When the latter is congenital, it is known as *albinism;* it may be generalized or localized. Complete albinism, in which there is a more or less total absence of pigment in the skin and its appendages, is a grave degeneration, very rare in the human race. Albinos have a waxy skin, white or very light colored hair on the scalp and body and a red or bluish iris.

Partial congenital achromia is the exact opposite of a pigmentary nevus; it is often familial, circumscribed in a nerve territory of only one side of the body, or it may consist of a few white spots, known as *achromic nevi.*

Acquired hypochromia is known as *leukoderma* and is usually secondary to a local process. It is frequently associated with hyperchromia at neighboring points, constituting the leukomelanodermas of vitiligo and some other analogous affections.

The *causes* of the dyschromias are extremely variable, and this pathogenic mechanism is far from being entirely understood. Among these causes, some act locally, while others are of a general kind, such as intoxications, infections, changes of the blood, nervous disturbances.

In both these cases, the pigmentation may assume the form of more or less circumscribed spots or it may become diffused over surfaces. The generalized melanodermas are always referable to a general cause. In the leukomelanodermas, the intervention of a nervous disturbance is usually admitted as essential.

For the *diagnosis* of a dyschromia it is first of all necessary to ascertain: if it is secondary to another process (macules) or primary or essential; if it is simple, without other changes of the integument, or associated with a dermatosis of different character; if it is circumscribed (pigmentary spots) or diffuse (melanoderma), generalized or regional.

The dyschromic syndromes to be discussed in the following have been arranged according to this plan; a paragraph has been added on the subject of vitiligo and other *leukomelanodermas*.

Finally, although these conditions are not due to pigmentary disturbances but to foreign bodies, a few lines are devoted to *tattoo-marks* and *argyria*.

ARTIFICIAL AND SECONDARY DYSCHROMIAS.

Artificial Pigmentations.—Any cutaneous irritation, especially when fairly strong and prolonged, may become the origin of an artificial local pigmentation. Certain individuals are evidently predisposed to it and its production is favored by stasis of the blood, as for example in varicose legs.

In some cases, the pigmentation is the direct and exclusive result of the irritation; in others it follows upon a hyperemia or even a bullous or eczematous process, etc., belonging in the last-named case to what I describe as macules.

Mechanical factors, friction by the clothing, corsets, shoes, bandages, repeated scratching in pruritus, give rise to hyperchromias of an often characteristic seat and configuration.

Among pigmentations due to physical factors, it suffices to call attention to the following: the tanned complexion produced by sunlight, the open air, electric light, the *x*-rays; the caloric pigmen-

tation of the face, forearms and trunk in blacksmiths, glass-blowers, bakers, etc.; of the thighs, in out-of-doors hucksters who have the habit of sitting on their foot-warmers and of any region of the body in persons who have abused hot compresses or very hot lotions for the relief of pain, pruritus, etc.; this is often arranged after the fashion of a wide-meshed network. [The dermatitis calorica of furnace men and others exposed to great heat is commonly followed by this reticular pigmentation.] Many chemical agents may cause very persistent pigmentations, even without a caustic action; and it is well for the physician to be forewarned, so as to avoid reproach under some conditions. This remark applies to nearly all rubefacients and counter-irritants, notably mustard plasters, methyl chloride, chloroform, tincture of iodin, etc. Applications of chrysarobin usually give rise to a bronzed erythema, followed by an extensive but temporary brownish pigmentation from which the healed patches of psoriasis stand out in white.

The *treatment* of the artificial pigmentations consists in the removal of the cause of the hyperchromia and in the employment of topical agents which will be discussed in connection with chloasma.

Macules.—I have for a long time been accustomed to reserve this name, which is popularly used interchangeably with the word spots, for the dyschromic but not cicatricial residues of a large number of cutaneous affections.

They follow upon excoriations, upon erythematous, vesicular, eczematous, bullous eruptions, such as burns, blisters from vesicants; purulent bullæ of impetigo; superficial folliculitides; the multiple lesions of scabies; papules of all kinds, etc.

They consist of a local, often distinctly circumscribed, pigmentation or sometimes of a central hypochromia surrounded by a pigmentary halo. They are often scaly at the onset and then of a perfectly normal surface.

In the absence of sufficient observation or information, maculæ may lead to numerous errors of diagnosis. It is necessary to guard against the confusion of maculæ, whose epidermis has its normal structure and which are always temporary, with *cicatrices* which are always permanent.

The seat, extent and configuration of the macules often possess important indicative value in regard to the causative dermatosis. The hyperchromic tendency of syphilitic papules of all kinds, in certain individuals, has long been emphasized. These *syphilides nigricantes*, to use A. Fournier's term, are sometimes a source of great distress for the patient.

A special group of pigmentary macules of hemorrhagic origin, in which the pigment is hemosiderin, is represented by the brown

spots following on traumatic or purpuric ecchymoses, hemorrhagic urticaria, varicose eczema, etc.

Under the name of *pigmentary dermatosis of the legs* (or *dermite jaune d'ocre*) have been separately described large more or less distinctly outlined spots of a uniform brown or mottled color, which are frequently observed on the legs of arteriosclerotic, varicose or diabetic individuals, or in Bright's disease, etc., and which persist indefinitely. They result from minute interstitial frequently repeated hemorrhages.

ASSOCIATED DYSCHROMIAS OR DYSCHROMIC DERMATOSES.

Several dermatoses give rise to a black or dark coloration of the integument which is not dyschromic, in so far as it results exclusively from an abnormal hue of the *horny layer*. In such cases, the colored layer can be finally detached by energetic scratching. This occurs in many hyperkeratoses and keratodermas, in ichthyosis niger, in psorospermosis follicularis, in certain seborrheas nigricantes, in pityriasis versicolor, in the carates [pinta], etc., and to a less degree, in kerosis.

Some dermatoses are actually pigmentary anomalies or may lead to such. Spots from antipyrin and from leprosy are, as a rule, erythemato-pigmented.

Various eruptions will be mentioned further on, which may be accompanied by a sort of pigmentary ataxia, such as lichen planus, various prurigos, etc.

Pigmentations also form an essential part of the pathological disturbances of acanthosis nigricans, xeroderma pigmentosum and several analogous cutaneous dystrophies; of v. Recklinghausen's disease and urticaria pigmentosa.

The reader is finally reminded of the pigmentary tumors, malignant, such as the nevo-carcinomata or pigmented sarcomas; or benign, such as pigmented nevi.

In this entire group of associated dyschromias, it is the underlying dermatosis, characterized by its peculiar lesions which establishes the diagnosis, governs the prognosis, determines the treatment and explains the pigmentation.

PIGMENTARY SPOTS.

Ephelides. — Ephelides, or *freckles*, confused by some dermatologists with lentigo, are small lenticular, rounded or oval, more rarely irregular spots, of a light yellow, café-au-lait or brownish color, perfectly flat, smooth and not scaly, generally isolated and numerous, or very profuse and agminated if not confluent, symmetrically arranged, having their seat of predilection on the face,

the nose, the prominences of the cheek bones, the forehead, the hands and forearms. More rarely, they are scattered over the shoulders, the arms, the legs, the buttocks and the genitals.

Ephelides are not congenital, but appear during childhood and youth, especially in blond or red-haired, anemic or lymphatic individuals and in persons enjoying excellent health.

Sunlight undoubtedly plays a considerable part in the mechanism of their onset, as suggested by their name; also, they are much more apparent during the spring and summer than in winter. But on the other hand, they are plainly hereditary or atavistic in certain families; they develop also in covered regions of the body and are in every way comparable to nevi.

The histology of ephelides shows merely an abnormal quantity of pigment in the basal epidermic cells, with presence of pigmentary cells in the papillary body.

Chloasma. — Chloasma uterinum consists of spreading spots, irregular in shape and outline, sometimes confluent in patches, of a yellow, brownish, or still darker color, situated almost invariably symmetrically on the forehead, the temples, the lateral portions of the cheeks and more rarely on the eyelids, the chin, or at other points of the body. The well-marked outline of chloasma distinguishes it from the caloric and solar pigmentations.

Chloasma ordinarily develops in *pregnant* women and persists until the reëstablishment of menstruation and often throughout life. At the same time, or even in its absence, there appears a pigmentation of the linea alba, the areola of the nipples and the vulva, especially in brunettes. Various pathological conditions, metritis, salpingitis, dysmenorrhea, etc., may produce identical pigmentations. Cases of chloasma without a demonstrable cause are also met with.

It seems probable to me that an irritation of the abdominal sympathetic nervous apparatus enters into its pathogenesis, as in the case of Addison's disease [and probably in acanthosis nigricans.]

The *treatment* of chloasma, like that of the ephelides and artificial pigmentations, yields no satisfactory results. Prophylactic measures would be the most advisable, with avoidance in predisposed persons, of cutaneous irritation or exposure to light; suitable treatment of uterine and abdominal affections or anemia and the lymphatic constitution when present. The administration of arsenic, which produces hyperchromia, is contra-indicated.

Locally, so-called decolorizing washes may be prescribed, applications of mercurial plasters during the night, or salicylated calomel pastes, or some peroxide cream. Exfoliation by means of exfoliating pastes is often successful in decolorizing the hyperchromic surfaces, but the cure is as a rule merely temporary.

Disseminated Pigmentary Spots.—As has just been shown, the ephelides and chloasma are regional affections. In the presence of disseminated yellow or brownish pigmentary spots, or a single spot, it is necessary to think in the first place of the artificial, macular and secondary pigmentations which have been discussed in the preceding paragraphs; next, of the pigmentary nevi and of v. Recklinghausen's disease, which includes some abortive forms, exclusively pigmentary; finally, one must keep in mind the possibility of circumscribed pigmentations in several melanodermas due to general causes.

The *blue* or shaded *spots* produced by the bite of phthirius inguinalis or the crab-louse, are characterized by their peculiar slate-blue coloring. They are of irregular form, of lenticular or nummular dimensions; the epidermis is in no way changed; they cause no itching and are of ephemeral duration [up to a week or more]. They are situated in variable numbers on the abdomen, the thighs, the back, and sometimes on the chest. The experiments of Duguet have shown them to be due to the local action of the venom of the parasite.

DIFFUSE DYCHROMIAS AND MELANODERMAS.

Diffuse pigmentations, whether generalized or, what is more common, regional or with regional predominance, are referable to: chronic infections, such as tuberculosis, syphilis, leprosy and pellagra; or to intoxications, such as arsenicism and phthiriasis; or to a variety of blood diseases; or to diseases of the nervous system. Occasionally, leukoderma is intermingled with hyperchromia.

Addisonian and Tuberculous Melanodermas.—In Addison's disease, the bronzed coloration of the integument is often delayed; but it may also precede by several years the other symptoms, such as asthenia, digestive disturbances and lumbar pains. The pigmentation, which is diffuse, of a red-brown or gray-brown color, first involves and stains the genital organs, the areolæ of the breasts, the articular folds and the uncovered portions, face and hands, and long-standing or recent scars. Jacquet has shown that a local irritation may "externalize" a latent tendency to pigmentation. On the hyperchromic surfaces, normal areas simulating leukodermic spots are sometimes seen.

Very commonly the pigmentation involves also the buccal mucosa, where tan or brownish spots, distinctly outlined or with diffuse margins, are noted on the cheeks, the lips, the tongue, the gums and the palate. [Pigmentation of the mucosa sometimes precedes that of the cutaneous surface.] The cutaneous and mucous lesions consist of an excess of melanin in the epidermis and cutis.

The melanoderma of Addison's disease is now known to be referable, not so much to a change of the suprarenal bodies themselves as to a lesion or irritation of the pericapsular sympathetic nerve apparatus which apparently presides over the regulation of the pigment. It has occurred to me that the pigmentations of acanthosis nigricans, of chloasma and perhaps that of pigmentary syphilides, may have an analogous pathogenesis.

The melanoderma of tuberculous patients has long been known, especially in cases of tuberculous peritonitis or enteritis. It consists of a dusky or brownish coloring of the genitals, the abdomen and sometimes the neck. It evidently results from the same conditions as genuine Addison's disease, from which it differs as a rule only by its more limited diffusion especially on uncovered parts.

Pigmentary Syphilides.—Aside from the posteruptive pigmentary macules referred to before and the tertiary leukomelanodermas to be discussed further on, syphilis very frequently produces a truly specific areolar pigmentation of the neck, which ranks among its most significant symptoms.

This areolar pigmentary syphilide is more common in women than in men. It appears as early as the second or third month, or in the course of the first year, rarely after two years and has a very indefinite duration which it is very difficult to determine [five to fourteen months, according to Jadassohn]. It consists of a grayish or brownish more or less dark hyperchromia, with diffuse borders, interspersed with islands of distinctly outlined white spots varying in size from a lentil to a franc, so that the whole forms a network with large strands, usually better marked on the lateral portions of the neck (Fig. 109). This "necklace of Venus" may send radiating processes on the chest, often in front of the axillæ, on the flanks, or still farther. In some cases, pigmentary macules are found in the center of a few of the white areolæ.

The pathogenesis of areolar syphilide is still a matter of controversy. By some, the pigmentation is interpreted as the primary and exclusive feature, the apparent decoloration of the meshes being referable to a contrast-effect; others assume true leukodermic patches, following upon a sometimes not very evident eruption, which become secondarily surrounded by hyperpigmented areas.

It is more than probable that both these interpretations are correct in different cases and that there are really two modes of formation of the pigmented network.

This dyschromia is almost pathognomonic. However, a few old observations are on record of areolar pigmentations of the same type referable to tuberculosis and chlorosis; these date back, it must be noted, to a period prior to the discovery of the serodiagnosis of syphilis.

Inversely, pigmentations of another type, analogous to chloasma, for example, have been noted in secondary syphilis.

The syphilitic hyperchromias are practically not amenable to specific treatment.

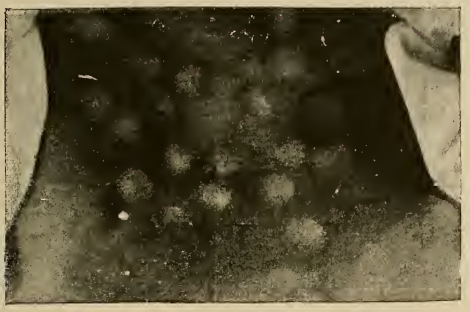


FIG. 109.—Areolar pigmentary syphilide of the neck. (After a cast in the Museum of the St. Louis Hospital.)

Dyschromias of Leprosy.—The initial erythemato-pigmented spots, leprous pemphigus, tubercles, infiltrations and ulcers often leave behind them hyperchromic or plainly achromic spots, or leukomelanodermas resulting from a combination of these two opposite disturbances.

For instance, white spots with a pigmented border may be observed—annular, band-like, or diffuse, or dark surfaces dotted with colorless spots, etc.

All these various manifestations, which were formerly designated under the names of *melas, leuke, morphea alba et nigra, vitiligo gravior,* are usually characterized by anesthesia.

The distribution of the pigment is variable. Hansen's bacilli are almost regularly demonstrable in sections of the skin, although in very small number.

Dyschromias of Nervous Diseases.—In the organic diseases of the nervous system, the disturbances of pigmentation are usually not pronounced.

In hemiplegia, cerebral tumors, infantile paralysis, progressive muscular atrophy, syringomyelia, tabes, the traumatic or toxic neuritides, etc., slight modifications in the coloring of the skin have been reported, associated with other trophic disturbances, hypertrichosis or alopecia, hyperidrosis, etc. The same observations have been made in mental diseases of the depressive or melancholic type.

On the contrary, the dyschromias are abundantly represented on the borderland of nervous pathology, in diseases placed here by their symptoms. In Raynaud's disease, in myxedema, in exophthalmic goitre, they are of frequent occurrence; vitiligo is rather common in Graves' disease.

In scleroderma under its different aspects, which can hardly be considered simply a nervous disease and in facial hemiatrophy, which is related to it in some respects, hyperchromia is a nearly constant symptom. A more or less deep, diffuse, areolar or spotted, primary or delayed pigmentation is noted, occupying the sclerotic areas, or the neighboring regions, or sometimes a large extent of the integument. Depigmentation of the sclerotic patches or regions is also not uncommon.

Dyschromias in Diseases of the Blood and in Cachexias.—It is enough to mention the hypochromia which may be seen in chlorosis, the chloro-anemias, pernicious anemia, the leukemias and in cancerous cachexia.

Inversely, it is not exceptional to observe various diffuse or regional melanodermas in these diseases more particularly in Banti's disease and in the pseudo-leukemias.

The melanoderma of bronzed diabetes, with or without pigmentary hypertrophic cirrhosis, is generalized, almost invariably spares the mucous membranes and consists of an infiltration into the cutis as well as other tissues, of an ochre-colored pigment known as rubigin.

In malarial cachexia, the coloration is ashy, a dirty gray, or yellow gray, diffusely and uniformly distributed. The malarial pigment, which is specific, is derived from the malaria-parasites, is carried to the skin by the blood (melanemia), and is here deposited, associated with an ochre pigment of hemic origin [hemosiderin].

Arsenical Dyschromias.—Arsenical melanoderma may develop independently of the age and sex of the patient or the nature of the arsenical remedy and its mode of introduction. Very minute doses are sometimes sufficient for its production, although as a rule, a prolonged absorption, medicinal, occupational, or accidental, is responsible.

The pigmentation may assume two forms which are sometimes combined; that of diffuse hyperchromia predominating in normally colored areas, cicatrices, or cutaneous regions which are subject to pressure, or that of pigmentary spots, which enlarge and become confluent. In patients who have been improperly treated with arsenic for psoriasis or lichen, a very deep generalized melanoderma is sometimes observed.

The coloration is iron-gray, bronzed, or even black. The uncovered regions remain relatively free, as well as the mucous membranes, with very rare exceptions.

Pediculous or Phthiriasic Melanoderma.—In paupers, tramps, scavengers and rag-pickers living in filth and misery, exposed to all sorts of vermin and whose clothing sometimes harbors incredible numbers of pediculi corporis, this condition is known to occur, so that the name of *Vagabond's disease* is usually entirely justified.

The pigmentation, of a dirty brown mottled with excoriations, crusts and cicatrices, predominates on the back, the nape of the neck, the shoulders, the waistline, and the thighs; but it may spread all over the integument, including the face and the extremities.

Thibierge has shown, and I was repeatedly enabled to verify the fact, that the pigmentation is observed even on the buccal mucosa, in the form of spots resembling those of Addison's disease.

The hyperchromia has been attributed to scratching, to bloody extravasations and to the local action of the venom of the lice. Its generalization and its possible localization in the mouth, show that this poison has a systemic action. The asthenia, the often very cachectic appearance of the patients, and the digestive disturbances from which they suffer, are additional proofs of this contention and help to render the differential diagnosis from Addison's disease very difficult in some cases.

VITILIGO.

Vitiligo is a non-congenital dyschromia characterized by the appearance of achromic or strongly hypochromic white spots, which are sharply limited and surrounded by a more or less extensive zone of hyperpigmentation.

Aside from the change in color, the skin presents no alteration of its surface, consistence or function.

The white spots of vitiligo have a milky or ivory hue, a dull sheen, a generally round, oval, or polylobular form, distinct faintly sinuous outlines. Sometimes they are in small numbers, but in other cases they may be so profuse and numerous as to cover a large portion or nearly the whole of the integument (Fig. 110).

The hyperchromia of the intermediate regions, which have a brown or grayish hue, is often particularly marked at the very border of the white spots; this arrangement conveys the impression of the pigment having been pushed out from the achromic surfaces. At the periphery of the hyperpigmented zones. the transition into the normal color is gradual and imperceptible, rarely marked by a distinct boundary.

The down and hairs of the white spots are either entirely colorless or of normal color.

The dyschromia is associated with no sensory disturbance, neither pain, nor itching, nor noteworthy anesthesia being demonstrable.

Jadassohn points out that the white spots are more susceptible

to solar erythema, while they are on the contrary more resistant to various cutaneous irritations and relatively immune to certain eruptions.

The *topography* of vitiligo is extremely variable, not infrequently its distribution is more or less symmetrical. It may occupy any region, although it shows a certain predilection for the back of the hands, the wrists and the forearms, the face and neck and for the genital organs and the neighboring areas. The mucosæ are always intact.

The *course* of the affection is governed by no rule; it may appear suddenly or more often insidiously; its extension is gradual and almost imperceptible or sometimes occurs in successive attacks.

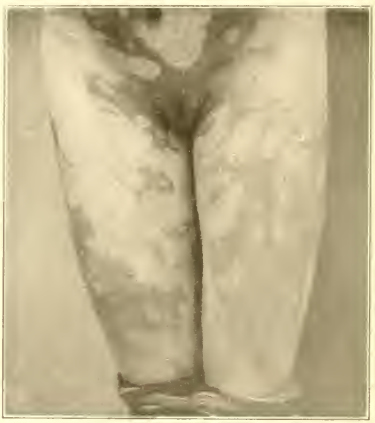


Fig. 110.—Vitiligo.

The dyschromia may remain almost stationary, with seasonal variations of its shades; more commonly, the white spots spread and become confluent in patches; they may even become generalized; but at the same time the degree of the hyperchromia diminishes. A complete cure is very rare.

The *etiology* of vitiligo is unknown. Adolescence, youth and the female sex seem to be relatively predisposing. It has been known to occur in persons who had been exposed to a nervous or emotional shock. Repeated traumatisms certainly play a part, for vitiligo is not rare at points exposed to friction as, for instance, from a hernial truss.

Moreover, it becomes associated too frequently for a merely

accidental combination, with alopecia, circumscribed prurigos, lichen planus, scleroderma and with a variety of nervous diseases, notably tabes dorsalis and exophthalmic goiter.

Like several writers, I have been struck with the relative frequency of syphilis in patients with vitiligo; however, there is at present no reason for admitting the existence of a syphilitic vitiligo, nor especially that all vitiligo depend upon this infection.

The *pathological anatomy* shows the almost complete disappearance of the pigment in the leukodermic spots and its abundance on the contrary in the epidermis and derma of the hyperchromic regions; but it affords no information as to the pathogenesis of these lesions.

The *diagnosis* is very easy in the vast majority of the cases. An even cursory examination suffices to guard against confusion with pityriasis versicolor and with the various pigmentations, circumscribed or diffuse, but without achromic spots, which have been mentioned in the course of this chapter. The light areolas of the pigmentary syphilide of the neck, which is moreover characterized by its seat, as well as those which may eventually be encountered in various melanodermas, are usually preserved normal skin areas and not leukodermas.

The only real difficulties which may present themselves are the following: In the course of leprosy, pigmented surfaces and achromic spots have been demonstrated and as a whole have been designated as vitiligo gravior; very evident sensory disturbances are present in these cases.

In certain families of temperate zones and more frequently in the colored races, congenital leukomelanodermas or cases of partial albinism have been noted; the dyschromia in these cases is absolutely stationary and sometimes symmetrical. So-called piebald negroes are probably in part referable to this congenital anomaly and in part to vitiligo or to leprosy.

The *treatment* of vitiligo usually is not very effective. In those cases where syphilis seems to be involved, or in the coexistence of tabes, mercurial treatment should be administered and I have obtained a remarkable success in one instance. It is to be feared that arsenic may aggravate the hyperchromia. As a rule, nothing can be done but to regulate the patient's general hygiene, to prescribe sedative or tonic hydrotherapy and electric treatments. Various opotherapies might be indicated in this affection. The overpigmented zones may be treated with the customary decolorizing agents.

Secondary Leukodermas and Leukomelanodermas.—These are distinguished from vitiligo by this essential characteristic that the dyschromia here is not simple, but accompanies an eruptive mani-

festation, or a change in the thickness, consistence and structure of the skin.

There is a coexisting eruption, with disturbances in pigmentation, in lichen planus, lichen hypertrophicus, circumscribed prurigo, etc.

There is sclerosis or atrophy of the skin in the following cases: the leukodermic spots of circumscribed scleroderma, also known as *morphœa nostras*, which are bordered by a lilac ring; the *morphœa* of *leprosy* which is anesthetic and contains Hansen's bacillus; the *cutaneous atrophies* in patches, often preceded by an erythematous stage; *lichen planus atrophicus* which has been papular; sclerotic *radiodermatitis*, which is interspersed with telangiectases.

Cicatrices, no matter of what origin, as well as linear atrophies are sometimes pigmented or leukodermic and in the latter case often surrounded by a zone of hyperchromia. This is of especially common occurrence in the cicatrices of tertiary syphilis. Under the name of *syphilitic leukomelanodermas*, extreme cases have been described, by A. Fournier, Gémy, etc., in which the skin finally becomes mottled with black spots, or sometimes with rounded or polycyclic white spots in the midst of pigmented surfaces, so as to simulate vitiligo from a distance.

TATTOO MARKS AND ARGYRIA.

Tattoo marks are spots on designs produced by the deliberate introduction of colored and insoluble particles into the cutis where they persist indefinitely.

These more or less artistic tattoo marks and inscriptions are usually made with lamp black or India ink when they are blue, or with vermilion when they are red; the colored powder is made to penetrate by means of closely bunched very fine needles. Tattoo marks are commonly seen in sailors, colonial soldiers, prostitutes and their cadets; but also occasionally in the educated classes, as the effect of a peculiar aberration.

Powder spots, resulting from a gunshot fired at close range and from the penetration of bits of charcoal, have a characteristic arrangement.

In certain occupations, among grinders, filers, stone-breakers, miners, etc., particles of steel, flint, or coal, may penetrate into the cutis and cause a sort of *occupational tattooing*.

Electrolysis, or hypodermic injections applied with steel needles, may likewise leave prints of *siderosis*.

Argyria is a slaty coloration, with bluish reflexes, which develops in persons exposed to the prolonged absorption of silver nitrate pills or other silver salts. It is generalized, but much more pro-

nounced on the face, the hands and the articular folds and may also affect the mucous membranes.

The silver granules, conveyed by the bloodstream, impregnate especially the elastic fiber and the capillaries, sparing the cellular elements.

Spots of local argyria may be encountered on the buccal, conjunctival and vulvar mucosæ, following upon energetic cauterizations with lunar caustic. [Very distressing cases of local and even quite extensive argyria have followed the use of the organic silver compounds in the treatment of dachryocystitis.]

Treatment.—In order to remove tattoo-marks, which is difficult, various caustic agents may be used. Variot recommends retattooing with a concentrated solution of tannin and then passing a silver nitrate pencil over the surface. A dry slough is formed, which brings the tattoo mark away with it, provided the slough is deep enough. I have advantageously employed crossed linear scarification followed by cauterization with pure carbolic acid. Applications of carbonic acid snow have also been recommended.

CHAPTER XVII.

CUTANEOUS ATROPHIES, SCLEROSES AND DYSTROPHIES.

Cutaneous atrophy is a nutritional disturbance of the skin in which there is a diminution in the number or volume of its constituents, the elastic tissue in particular; clinically, it manifests itself as a diminution of the actual thickness or consistence of the integument. The atrophic skin is therefore more supple, more easily folded and often thinner than the normal skin, its color is usually altered, either pinkish or of a pearly white. Sometimes, as in certain linear atrophies, for instance, the skin seems to be thickened on inspection, while remaining soft, depressible and easily folded; this depends upon its relaxation and infiltration by plasma, or upon the atrophied portion being pushed out, as it were, by the tension of the normal and more resistant integument of the vicinity (see Fig. 111).

Cutaneous sclerosis is a condensation of the skin-components, which may or may not be increased in size and number, but are always more heaped up and move less readily over each other. The sclerotic skin may therefore be thickened or normal or even thinned; in the latter case it seems to be atrophic; but it is always more firm, less depressible, generally difficult to fold and often it is adherent to the subjacent tissues.

Although *atrophoderma* and *dermatosclerosis* constitute different and in some respects opposite conditions, they are sometimes difficult to distinguish clinically; they are often combined or associated or follow one another, so that it is advisable to study the syndromes in which they are met with in the same chapter.

Finally, there exist alterations of the skin which can hardly be designated otherwise than as *cutaneous dystrophies*. The integument here is sometimes thinned, in other cases swollen, while its consistence is usually diminished. They form a natural group which logically takes its place by the side of the preceding.

The *pathogenesis* of these atrophies, scleroses, and dystrophies is sometimes evident, often on the contrary very obscure and certainly not uniform. Sometimes they are *congenital* and represent actual malformations (example: atrophic nevi, xeroderma pigmentosum); in other cases they behave like *degenerations* due to old age, repeated exogenic or endogenic irritations; again, they are *secondary* to a more or less definite inflammatory or neoplastic

process, of which they constitute a necessary or possible residue; or finally they seem to be *primary* or *idiopathic*, supervening without apparent cause and without being preceded by demonstrable lesions, so that their etiology and their mechanism altogether escape us.

Sometimes a relation may be observed between the dermatosis and some disturbances of the general health, but this is entirely absent in other cases.

Sclerotic and Atrophic Dermatoses.—The reasons for studying these together have just been stated. Practically, the two are inseparable. My object here is not to establish absolutely logical groups from the standpoint of general pathology, but to present clinical pictures conforming as closely as possible to actual facts.

The atrophies and scleroses are deuteropathic or idiopathic.

1. The *deuteropathic*, generally diffuse forms, with or without sclerotic retraction of the skin, following upon various grave dermatoses—pityriasis rubra of Hebra, malignant herpetides, congenital pemphigus, some cases of pemphigus foliaceus—will not be commented on. The atrophy here is merely an epiphenomenon and the dermatoses in the course of which they occur have been described elsewhere (Chapters VI and X).

2. Other scleroses or atrophies, likewise deuteropathic, are entitled on the contrary to special consideration; they are sequelæ of lesions or of circumscribed dermatoses, persisting long after the causative affection, which can sometimes be retrospectively recognized through them. These are the *cicatrices* and *cicatricial atrophies*.

3. Next to them I place the *linear* or *macular atrophies*, a heterogeneous group of atrophies of variable pathogenesis, comprising protopathic forms and others which are deuteropathic.

4. A description of the *idiopathic atrophies* will follow. These curious and interesting affections are as yet imperfectly defined and their etiology is unknown. A distinction is made between diffuse or regional forms and other macular and disseminated forms.

5. *Congenital atrophies* are very rare. A generalized form has been observed in degenerates, the offspring of parents tainted by alcoholism, tuberculosis, syphilis, etc. Their integument is thin, smooth, allowing the vessels of the hypoderm to shine through, pale or variably pigmented, and extremely vulnerable. Partial congenital atrophies, or atrophic nevi, are more or less distinctly outlined spots of the same appearance, or yellowish and prominent through hypertrophy of the hypoderm; they may assume a zoniform arrangement.

6. I shall finally discuss the *sclerodermas*, diffuse and circumscribed scleroses in the true sense of the term and which are still regarded as idiopathic.

7. In another paragraph will be united a few *regional dermatosscleroses* which notwithstanding the analogy of their lesions, a mixture of atrophy and sclerosis, differ from each other by their localization, their course and probably by their pathogenesis.

Cutaneous Dystrophics.—This denomination would fit a considerable number of chronic dermatoses: Cutaneous atrophies, scleroses and hypertrophies, many keratoses, dyschromias, folliculosis, trichoses and onychoses, etc., really resulting from a nutritional disturbance of the tissues of the skin. I shall limit myself to grouping under this heading: *xeroderma pigmentosum, senile degeneration, presenile dystrophy,* and finally two rare degenerations: *pseudo-xanthoma* and *colloid milium.*

CICATRICES.

A cicatrix consists of newly developed tissue which has replaced a loss of substance or followed an inflammatory process. This new tissue is always fibrous; moreover, the reconstruction of the skin always remains imperfect for there is a lack of elastic tissue, smooth muscle fibers, hairs and cutaneous glands, often even of the papillary body. A cicatrix may therefore be said to be at the same time a deuteropathic dermatosclerosis and a cutaneous atrophy, even when the scar itself is hypertrophic.

A *good* cicatrix is smooth, level or slightly depressed, pink or white in color, supple and movable on the underlying tissues. A *vicious* cicatrix is uneven, salient or furrowed by retracted bands; a *keloid* cicatrix is the seat of a prominent and hard fibrous hypertrophy.

Very superficial cicatrices are recognized only by a very trifling depression and a somewhat glistening sheen, with an altered "grain" of the skin, sometimes with dilatation of the follicular orifices; these changes being absent in simple macules.

Thicker cicatrices are at first rose-colored, ultimately white or pigmented, sometimes scaly and usually firm on touch. Being less extensible, less elastic than the healthy skin and often provided with a less active blood supply, they are liable to become the seat of tears and cracks, or they may be so highly vulnerable that slight traumatisms may give rise to serous or hemorrhagic bullæ or maintain ulcers in the scars; sometimes they are tender or even spontaneously painful. It is readily understood that when the causative lesions have involved the deep parts, bones, muscles, tendons, etc., the cicatrices may be *adherent.*

The *origin* of cicatrices is extremely variable. From this viewpoint they may be divided into three groups:

1. Those which are due to an artificial loss of substances, trauma-

tism, wounds, accidental or surgical incision; a caustic agent, a burn, etc.

2. Those which result from some ulceration.

3. Those which are derived from an interstitial pathological process without apparent loss of substance, whose repair has given rise to sclerosis with a marked change in the structure of the skin. Between cicatrices of this kind and what I shall describe as *cicatricial atrophies*, it is not really possible to trace a distinct border-line.

Although no cicatrix is strictly pathognomonic in itself, its probable origin can often be surmised; and scars being indelible constitute stigmata of special value.

In estimating the symptomatic significance of cicatrices, it is necessary to keep in mind especially their extent, their number (variola, acne), their configuration (syphilis, tuberculosis), their seat (bubos, chancroids, chancres, lupus, etc.), their depth (ulcers, etc.), and even their color (favus, syphilis, etc.). It must be remembered, however, that their features may be modified by various accessory factors, superadded infections, faulty dressings and an unfavorable general, regional or local territory.

Among the pustular and ulcerative affections which leave cicatrices, I shall mention variola, ecthyma, pustular acne, acne necrotica, furuncle, carbuncle, ulcerative zona, soft chancre; the multiplicity of cicatrices of this type, their small extent and their localization are more or less plainly characteristic.

All tubercles, syphilitic, tuberculous or leprous, are almost necessarily followed by cicatrices; and this fact is implied in the definition of these lesions as *non-resolutive*.

For simplicity's sake, I shall proceed to sum up the features of the cicatrices caused by these three great infections, independently of the original lesion.

In syphilis, the chancre leaves a cicatrix only when it has ulcerated; actually, in nearly one-half of the cases.

Ulcerative secondary syphilides may spatter the integument with more or less deep, flat or honeycombed cicatrices, often with a pigmented border.

The cicatrices of ulcerative or gummous tertiary syphilides are said to be characteristic on account of their white and smooth appearance with a zone of peripheral pigmentation. In reality, it is their configuration and arrangement which are of special diagnostic value; they generally have sharp and regular contours, orbicular or semicircular, or are made up of disks arranged in arcades, or of reniform polycyclic patches. The cicatrices of tubercular syphilides are often of irregular surface, honeycombed, purplish or brownish, checkered with white stars.

The presence of round and very superficial cicatrices on the

buttocks may be cited in favor of a diagnosis of congenital syphilis; but their value would be very small in the absence of other stigmata. Radiating cicatrices of the lips are on the contrary very characteristic in themselves.

The cicatrices of ulcerated tuberculosis, bony, articular or glandular, as well as those of tuberculous gummas and ulcers, are often distinguished by their irregular, sinuous and eroded contours, and their uneven surface, which presents retracted strands, promontories and bridges. The latter—consisting of minute strands adherent only by their extremities (under which horny scales, colored black by dust, accumulate) and which can be raised by slipping a pin underneath—possess, I believe a real diagnostic value for tuberculosis; but they are not absolutely pathognomonic, for I have seen "bridged" cicatrices as a sequel of sporotrichotic lesions, of carbuncle in a diabetic patient, etc.

Lupus leaves very variable cicatrices, according to its varieties; flat, white and smooth in its erythematous resolving form; thicker, often rose-red, sometimes vicious and keloidal, in the deep forms. They may cause serious deformities of the orifices of the face, for example ectropion, stricture or atresia of the nares and the mouth; adhesions of the fingers or toes (Fig. 170), contractions of the joints, etc. They are frequently the seat of renewed growth of lupus nodules.

Leprosy furnishes very variable cicatrices, superficial or deep, supple or very sclerotic, prominent or depressed, following upon bullæ, leprides and especially upon tubercles and infiltrations; they are often described as *morphœa leprosa;* they are white, or sometimes deeply pigmented and are characterized by their anesthesia. Aside from these morpheas, mention must be made of the deep cicatrices left by the ulcers of leprosy and the diffuse dermatosclerosis of the extremities in the mutilating form.

The vegetative dermatoses—such as, Oriental boil, yaws, pemphigus vegetans, iodide and bromide eruptions, etc.—also leave cicatrices, often irregular, with pigmented borders. Needless to say, this is likewise true for ulcers of all kinds, phagedenas, gangrenes; the scars which follow depend in the extent, configuration and depth of the original lesions.

The same remark applies to tumors, which may give rise to cicatrices, benign tumors like certain nevi mollusci or absorptive angiomas and malignant epitheliomatous tumors. In this way, secondary carcinomas of the skin, ulcerated or not, of the type formerly named scirrhus, become the seat of a fibrous thickening which appears exactly like a lardaceous scleroderma. Finally, in cutaneous epithelioma of the variety known as *flat cicatricial epithelioma,* the center of the patch is often seen to become sclerotic,

the epithelial newformation remaining demonstrable only at the borders in the form of a narrow beaded margin.

It is useful to know that *vice versa* cicatrices may become the starting-point of epithelioma which is generally of the lobulated type.

The diagnosis of the origin of cicatrices is often greatly helped by the history, by serodiagnosis and by the general examination of the patient.

Cicatricial Atrophies.—Alongside of these often irregular and important cicatrices which have just been discussed, must be placed more discrete, usually macular lesions, of atrophic appearance in the clinical sense of the word. The name of *cicatricial atrophies* is reserved for these lesions.

Cicatricial atrophy, generally in the form of white, flat, smooth, more or less indurated patches, is the inevitable result of lupus erythematodes discoides, the different forms of depilating folliculitides, and Brocq's pseudo-alopecia; of the resolutive tuberculous lupus; tubercular syphilides and the majority of non-ulcerating lepromas.

It characterizes a variety of lichen, known as *lichen planus atrophicus*.

Several bullous affections, notably pemphigus congenitalis, leprous pemphigus, rarely Duhring's disease, leave spots or patches of thinned skin, often pigmented or purplish, somewhat indistinctly outlined.

Small atrophic and leukodermic spots, associated with pigmentations, are noted in various dystrophies, such as xeroderma pigmentosum, senile degeneration, etc.

Keratosis pilaris, on healing, leaves behind it a crop of punctiform cicatrices, the site of which is typical.

Favus very often produces large, smooth, bald and cicatricial surfaces, usually of a pinkish color, but sometimes decidedly white.

Mention must also be made of radiodermatitis, which even in the absence of ulceration, may be followed by white cicatricial atrophies with diffuse borders, mottled by telangiectases and pigmentary spots.

The fact that certain erythematous dermatoses terminate in cicatricial atrophy, led Unna to establish his group of *ulerythema* (from οὐλή = cicatrix), comprising a centrifugal type, lupus erythematodes, and ophryogenic and sycosiform types.

This atrophy-producing tendency belongs also to certain erythematous tuberculides. Finally, some special cases of erythema, urticaria and purpura, have been known to terminate in cicatricial atrophies, which have been compared with the idiopathic macular atrophies.

The *diagnosis* of the origin and nature of these cicatricial atrophies

generally rests upon their form, their dimensions, their number, their topographical seat; sometimes, it has been possible to follow their development, or the original lesions can be discovered, either at the circumference of the spots or elsewhere.

Summarizing, the clinician when confronted with an atrophic or sclerotic spot must think in the first place of a cicatrix and inquire as to its origin; next, if there is no genuine cicatrix, he must think of the cicatricial atrophies; the processes which may terminate in a lesion of this nature have just been shown to be extremely numerous and different; all possible theories should be reviewed; third, when all the preceding conditions can be excluded, it is justifiable to assume an idiopathic macular atrophy.

Pathological Anatomy.—The pathological anatomy of cicatrices varies according to the depth and character of the lesions which have caused them. The cutis, or at least the papillary body, must have been involved, for otherwise repair would have occurred without a scar.

In a general way, their structure is as follows: the epidermis is more or less thick, often hyperkeratotic or parakeratotic, smooth, or sends out a few irregular proliferations at its deep aspect, it covers a dense fibrous tissue, composed of parallel connective-tissue bundles, deprived of elastic fibers or at least of a regular elastic network.

The papillæ and the entire papillary body are often missing. In the spaces between the fibrous tissue rows of embryonic or plasma cells and often very numerous mast-cells persist for a long time. The vessels, relatively scanty, but often telangiectatic under the epidermis, follow variable paths, having nothing in common with the normal vascular distribution. Not infrequently, there are collections of pigment in the gaps of the fibrous tissue adjacent to the epidermis, in the area or at the circumference of the cicatrix; more rarely, pigment is noted in the epidermis itself.

The hairs and pilosebaceous follicles, as well as the sweat-glands, are absent or sometimes transformed into milium cysts.

Treatment.—Cicatrices may, in the course of time, become flexible and resume a nearly normal color; but they are never entirely obliterated.

The prophylactic treatment of vicious cicatrices consists in the use of proper dressings, autoplastic operations: the so-called epidermic grafts of Reverdin, or dermo-epidermic grafts of Ollier-Thiersch, or the small deep skin grafts of American writers may be indicated in cases of extensive loss of substance.

Sometimes, when circumstances are favorable, it may be advantageous to excise a disfiguring cicatrix, in order to replace it by a less evident linear scar. Local massage, sometimes scarifications, often the application of mercurial ointments or radiotherapy, may

improve an unsightly cicatrix. Injections of thiosinamin or fibrolysin are not devoid of danger and yield few durable results. As regards keloids, this subject will be discussed further on.

By means of ionization of a solution of potassium iodide on the negative electrode, it is possible to liberate, make supple and bleach vicious cicatrices, even in longstanding cases (Chiray and Bourguignon.)

LINEAR AND MACULAR ATROPHIES.

Linear atrophies—called "vergetures" in French, from their resemblance to the livid streaks left on the skin by blows with a strap or rod—are also known as striæ atrophicæ, striæ gravidarum, striæ distensæ, etc.

These are cutaneous atrophies of elongated form, prominent, level or depressed, but always soft and indentable—which seem to to be due to overstretching of the skin; they are indelible, but often become less visible in the course of time.

Linear atrophies have a length of one to several centimeters, a width of 1 to 10 mm., or more, their form is elongated, spindle-shaped and often undulating. Their color, of a dusky or bluish red when they are recent, frequently passes into a pearly white; sometimes, on the contrary, they are brownish. Their borders are distinct; their surface is smooth, or puckered, or designed in large lozenges; on touch they give a sensation of softness, of relative emptiness; as if a very thin skin were resting on a soft and yielding tissue.

Almost invariably multiple and usually symmetrical, these linear atrophies may develop in many regions, principally on the abdomen, but also on the thighs, the loins, above the knees, on the flanks, the breasts, the buttocks, etc. Their direction corresponds as a rule to what are known as the lines of cleavage of the skin; their long axis is perpendicular to the direction in which the maximum tension which seems to have caused them has taken place; they are usually vertical on the abdomen, the trochanteric and deltoid regions; transverse on the flanks, the loins and above the patella; radiating on the breasts.

Linear atrophies are much more common in women, even outside of pregnancy (36 in 100 cases in adult women, against 6 per cent. in men, according to Schultze); although pregnancy is the most common cause. They are observed in nine-tenths of all pregnant women though some women never acquire them, even after ten to fifteen confinements. Among other frequent causes of linear atrophies must be mentioned obesity and typhoid fever.

In an endeavor to ascribe a mechanical pathogenesis to these lesions—a gradual or a rapid distention of the skin which plays an

evident but not exclusive part in their production—attention has been called to the effects of growth, anasarca, voluminous tumors, or traumatisms.

It is certain, however, that another mechanism intervenes aside from this distention. As a matter of fact, linear atrophies may be absent in cases of enormous ascites or very large hernias; in all probability, there is no actual distention of the skin in corpulent or in rapidly growing individuals; linear atrophies may occur even as a sequel of emaciation, in typhoid fever, tuberculosis, other severe infections and certain nervous diseases.* I have seen linear atrophies and postsyphilitic atrophies coexisting in the patient shown in Fig. 111, who had lost weight, from 108 to 72 kilos. There is accordingly reason to suspect, in affected individuals, the existence of a special delicacy of the skin, notably its elastic tissue, either of congenital or toxi-infectious origin.

The *pathological anatomy* of linear atrophies sufficiently explains their clinical features. The epidermis and the papillary body are spread out or folded, the connective-tissue bundles of the cutis are parallel and atrophied.

The fundamental lesion consists of the disappearance of the elastic tissue network, the retracted and shrivelled stumps of which are seen on either side of the lesion; it is noteworthy that this rupture is not accompanied by a demonstrable degeneration, such as a transformation in elacin of Unna, etc.

Linear atrophies show no tendency to repair. No treatment can guarantee their disappearance, although they may be rendered less apparent by hygiene, hydrotherapy, tonics. It is doubtful if supporting appliances, pregnancy belts and so forth, are of any use as preventives, but their employment should nevertheless not be neglected.

Round or Macular Atrophies.—This name, as well as *maculæ atrophicæ* or *postsyphilitic atrophies*, designates lesions which are entirely analogous, except in form, with linear atrophies. These spots are depressed, level, or prominent, smooth, shrivelled, or pitted, according to the state of tension or relaxation of the skin, lavender or white according to their age and always very soft and depressible. They are round or oval, punctiform or lenticular and are scattered without order, usually in large numbers on the flanks, the chest, the back, or the shoulders (Fig. 111).

The relation of round atrophies to syphilis is undoubted; they belong to the secondary stage, sometimes associated with an eruption of papular syphilides or with the pigmented syphilide of the neck. Their development could sometimes be followed as a sequel and at the site of lenticular papules. There are cases, however, in which the preëxistence of lenticular papules, or even of roscolar

spots, at the atrophic points cannot be demonstrated and is positively denied by the patient.

The structure is the same as that of linear atrophies and the treatment is equally futile.

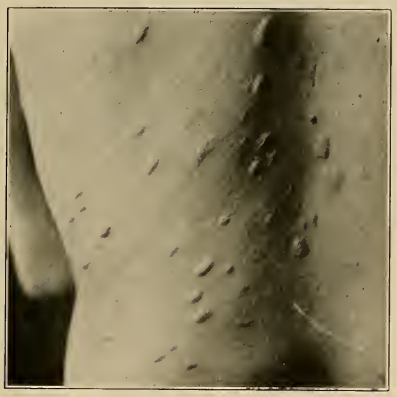


FIG. 111.—Round syphilitic atrophies of the dorso-lumbar region; their elevation, very apparent in extension of the trunk, entirely disappeared when the trunk was flexed or the skin stretched. The patient showed, in addition, linear atrophies on the abdomen and the hips.

IDIOPATHIC ATROPHIES.

The qualification "idiopathic," it is needless to state, merely expresses our ignorance as to the underlying causes. In regard to the pathogenesis of these atrophies, although they may sometimes appear primary, they are generally supposed to be the result of a preliminary or concomitant inflammatory process. Cases can be distinguished in which this process is clinically evident and others in which it is not demonstrable; but it is probable that even in the latter an inflammation is present in the first stages of the affection and could be revealed by histological examination. On this view are based the terms *dermatitis atrophicans*, *erythema atrophicans*, etc., which have been suggested. It can by no means be claimed that the group of idiopathic atrophies presents a unit, the contrary being probably true. However that may be, from the morphological point of view, they may be divided into two groups, according to their being diffuse or circumscribed.

1. **Diffuse Idiopathic Atrophies.**—The first observations were made by Buchwald (1883), Touton and Pospelow. Ten years later, F. J. Pick described a type of this affection under the name of *erythro-*

media and Herxheimer in his turn, with Hartmann, resumed its study, under the name of *acrodermatitis atrophicans chronica*. The affection is progressive and develops in interrupted attacks; but in the cases of Kaposi and of Colombini its course was rapid. Its seat of predilection is the limbs, but it may reach also, or independently, various regions of the trunk, the hips and even the head. The most distinctly marked clinical type is described in the following:

Erythromelia of Pick, or *acrodermatitis chronica atrophicans* of Herxheimer.—In this form the dermatosis attacks first the extremities, more particularly the dorsal aspect of the hands and feet and the extensor surfaces of the elbows and knees. It sometimes seems to progress from the periphery toward the center, but usually settles or markedly predominates in the above enumerated regions. The thighs and arms are rarely involved, the shoulders and hips only exceptionally.

The lesion consists of an atrophy which may seem primary; in other cases, it is preceded by a red edematous infiltration of firm consistence; from the hands and feet, this sclero-edematous infiltration, which deforms the fingers and toes and impedes their movements, may invade the forearms and legs, often in the form of a pre-ulnar and pre-tibial band with redness in the vicinity.

The atrophy, primary or following this inflammatory stage, is red or rose-colored, slowly extensive and permanent. The thinned-out skin, through which can be plainly seen the venous network and the tendons, is nearly alopecic, slightly squamous and puckers like tissue-paper; on touch, it gives an impression of softness, like that of moist chamois-skin.

Histology shows, aside from a diminution of the elastic plexus, an edematous and cellular infiltration containing numerous plasmocytes.

Erythromelia more frequently attacks men of mature age than women. It lasts for years. Its ultimate state is not known; perhaps a few cases terminate in recovery.

When it is limited to the hands, it might be confused with pellagrous erythema; or in its very extensive diffuse form, with senile atrophy. The presence of an edematous inflammatory infiltration at certain points suffices for the avoidance of these errors. Redness and atrophy serve to differentiate erythromelia from scleroderma at the onset, where the fingers are likewise stiff and infiltrated; but there are cases in which the two processes are combined.

In the *poikiloderma vasculare atrophicans* of Jacobi (1909), or *reticular atrophy* of Zinsser, the plexiform atrophoderma is combined with telangiectasis and pigmentation.

2. **Macular Idiopathic Atrophies.**—This second group of cases was established about the same time and parallel with the first, although

with less certainty. A few old cases of cyanotic macules (Besnier and Fournier) and of erythematous atrophy in patches with peripheral extension, etc., were known; but this clinical type had no scientific standing until the publication (1891) of Jadassohn's case of *anetoderma erythematosum*, or *atrophia maculosa cutis*.

Galewski, Nielsen, Heuss and others have contributed case-reports; the observation of Thibierge (erythematous atrophoderma) approaches it in certain respects, while also offering points of contact with lupus erythematodes. But the cases of Pellizari (urticarial erythema), Balzer (erythema polymorphe atrophicans), Hallopeau (chronic urticaria with cicatrices), Pospelow (purpura atrophicans), Nikolsky, etc., must according to Heuss be transferred from the group of idiopathic macular atrophies to that of cicatricial atrophies.

It has been asserted that macular atrophy represents merely a special case, a circumscribed form, of idiopathic atrophy. Certain observations, due to Herxheimer, Thimm and others, seem to establish a transition between the two classes. The objective differences are, however, as a rule, very marked, as appears from the following description.

Macular Atrophy or Anetoderma Erythematosum of Jadassohn.— There is a scattered, more or less profuse eruption of atrophic spots, sometimes predominating on the extensive surface of the limbs, on the flanks and on the back. Most commonly, they are nummular and rounded, sometimes irregular or even in striæ; their color varies from purplish red to pearly white; their contours are distinctly marked by the difference in color and by the depression of the skin at their site; on the least movement, the epidermis on their surface becomes folded. The integument as a whole has a very peculiar doughy and soft consistence; on touch, the spots convey the impression of holes dug in the skin; their flabbiness contrasts with the firm consistence of the atropho-sclerotic spots described in the next section.

Jadassohn believes he has shown that at the onset the lesion is a dermic papule analogous to a syphilitic papule; others have seen spots appearing atrophic from the start. They progress slowly for several weeks or several months; sometimes a rose-colored circle marks the zone of invasion; it is said that some spots may completely disappear.

Nearly all the reported cases were observed in young females who often were tainted with tuberculosis.

The differential diagnosis must be made from nevi atrophici, from cicatrices and from cicatricial atrophies. I have observed spots of identical appearance in Recklinghausen's disease, the true character of which could only be established by histological examination.

SCLEROTIC ATROPHIES IN SPOTS.

In juxtaposition to the soft macular atrophy, or anetoderma, a place must be assigned to a clinical picture to which attention has been drawn by many recent publications. It consists of both atrophic and sclerotic spots, of a glistening, pearly or porcelain white, level or slightly depressed, *firm on touch*, which appear in variable number in certain regions. Their dimensions vary in the same case from a pin-head to a silver quarter, but they may become confluent in larger spots.

It is more than probable that this syndrome does not constitute a single affection. American and English writers designate it under the name of *White spot disease*, first employed in 1903 by Johnston and Sherwell. It may be brought about either by lichen planus sclerotiens or by morphea guttata. In a given case, an effort must be made to differentiate between these two affections, on the basis of what will follow. This is often impossible and the question then arises if there is not another pathological entity of nearly identical appearance.

Lichen planus scleroticus vel atrophicus (Fig. 35) is met with in both sexes and may be situated in any region of the body, but especially at the nape of the neck and on the wrists; the small white spots, finely criss-crossed, not frequently studded with horny plugs, may have a slightly raised border; they begin as polygonal papules of tawny hue and accordingly represent cicatricial atrophies. When there is a coexisting ordinary lichen planus on the integument or on the buccal mucosa, this of course settles the diagnosis. The histology of the lesions shows a small patch of subepidermic sclerotic atrophy, under which may be found, in recent lesions, a remnant of the circumscribed cellular infiltration belonging to lichen planus. The *lichen albus* of Zumbrusch, be it stated, is nothing other than this sclerotic lichen planus.

Morphœa guttata, or **superficial circumscribed scleroderma**, or **parchment-like scleroderma** (**"Kartenblattaehnliche morphoea,"** of Unna) or **white-spot disease**, properly so called, is observed rather in young girls or women of any age, more or less tainted with tuberculosis; its seat of predilection is on the upper chest, the shoulders, the neck, the nape of the neck and the perigenital region. The lesions which are lenticular or nummular with a tendency to depression, are of a pearly or porcelain-white color and are bordered by a narrow pink or purplish zone; they may bear a few thinly scattered horny plugs; the sclero-atrophic spots originate under this form and may in all probability disappear; their number is very variable.

The condition represents a slight hyperkeratosis, with a patch of compact thickening in the cutis of modified staining capacity,

poor in cellular elements; the preliminary pathological process is unknown.

Several cases of white spots coexisting with scleroderma in patches or in bands have been published; however, the relationship between these two affections is not certain for all the cases, some authors have thought of a relation with lupus erythematodes.

Treatment by means of radiotherapy, electrolysis and thyroid extracts, seemed to be beneficial in several cases.

SCLERODERMA.

In the very extensive realm of the dermatoscleroses, the sclerodermata form a limited group which comprises apparently idiopathic affections, meaning that their nature is entirely unknown. They are divided into four groups:

Sclerema Neonatorum.—A child, born apparently quite healthy, may present at the end of a few hours, or after two to ten days, sometimes still later, a progressive induration of the integument, constituting sclerema.

The affection, which is very rare, begins at the posterior portion of the lower limbs, reaching the loins, the back and the entire body; it may also begin in the face.

The skin, of a yellowish, livid, or lilac white, is not depressible with the finger, as in edema, but firm and cannot be folded. Movements are impeded, the infant is unable to take the breast, emaciation is rapid, respiration is interfered with, the pulse is slowed and death is apt to supervene in three or four days, almost invariably with hypothermia or in convulsions.

This disease, which presents the behavior of an infectious process of indefinite character, differs altogether from congenital generalized cutaneous atrophy. It has likewise nothing in common with fetal ichthyosis, in which the integument is red, tense and covered with a carapace of scales.

For the *treatment* of sclerema, the children must be kept warm in the incubator and fed with a stomach-tube, if necessary. Cures have been observed to follow mercurial inunctions.

Generalized Scleroderma.—This disease, also named *edematous scleroderma* (A. Hardy), or *sclerema of adults*, or *sclerema* (Besnier), manifests itself in two forms:

In the *acute form*, the onset is sudden and the course is rapid. The patient complains of stiffness, impairment of movements of the trunk and limbs; breathing becomes difficult, the integument becomes thickened and diffusely indurated. This form is very rare and often fatal in a few weeks, or at most a few months.

The *slow form*, likewise uncommon, is preceded by prodromata:

disturbances of the general health, emaciation, febrile attacks, neuralgias and arthralgias, stiffening of the limbs, heat and itching in various portions of the skin, sometimes with erythema, or local syncope, or edema, or secretory disturbances, hyperidrosis and so forth.

Next follows a sclero-edematous period, during which the integument of the entire body and the limbs or of extensive areas becomes thickened, infiltrated with a sort of lardaceous, non-depressible edema, with adhesion to the subjacent tissues; so that it is indurated, tense and cannot be folded. The color is a yellowish white, interspersed with gray or brown or lilac spots. The face becomes like marble, its folds disappear, movements of the eyelids, forehead and lips become impossible; speech and the ingestion of food are hampered. The induration of the neck and the chest interfere with respiration, sometimes with swallowing. The limbs are stiff or actually immobilized, more seriously affected in their first segments than at the extremities. The boundaries of the affected regions are always diffuse.

The sclero-atrophic period gradually follows upon the preceding, usually after a few months. The skin becomes fibrous, retracted, and adherent to the muscles and the bones; the subcutaneous panniculus disappears; the muscles are themselves sclerotic and movements are furthermore impeded by fibrous bands. The mucous membranes may be involved. The patients are enormously inconvenienced by the cuirass in which they are enclosed and they complain of persistent cold; sensibility is preserved.

Sclerema supervenes as the sequel of a fall into cold water, a severe traumatism, or an infectious disease. Its course is in attacks, with remissions; death results most commonly from pulmonary, digestive or renal complications, or through cachexia.

Progressive Scleroderma, Sclerodactylia.—The onset here is on the upper extremities, rarely in the face. This form is systematized, symmetrical and progressive.

The first *symptoms* consist of nervous and vascular disturbances, numbness, sensations of cold, spasms, shooting pains, local asphyxia or local syncope; they occur in attacks, as in Raynaud's disease, or may be constant; occasionally hyperidrosis or pemphigoid bullæ have been noted. These phenomena may be continued during months and years. Asphyxia of the nose and ears, and paresthesias of the face, have also been observed.

In the fully developed stage, which supervenes at the end of several months or years, the fingers become pointed, their skin is thinned and adherent to the bones; they appear hard and dry, can no longer be flexed or extended and are of a grayish or slightly livid color. The process begins at the last phalanges, reaching the root of the

fingers, the hands, the forearms, etc. The fingers transformed into rigid fusiform sticks may become the seat of sluggish ulcers or of gangrenes, or bony disintegration; terminating in mutilations like those of leprosy. The nails are raised, thinned, or onychogryphotic. The hypoderm, the muscles and tendons participate in the sclerotic induration, thereby leading to a real mummification.

Entirely analogous, although usually less pronounced alterations occur in the lower extremities (Fig. 112), the toes become turned out-

Fig. 112.—Progressive scleroderma.

ward; ulcerations and a certain degree of plantar keratoderma are not uncommon.

In the face, the appearance is characteristic; even more so than in generalized scleroderma.

The wrinkles and folds are obliterated, the features are fixed and immovable; the ears are rigid, the nose is pointed, the lips are thin and tense, the eyelids cannot be completely closed, mastication and deglutition are impeded; the tongue may become atrophied. I have seen aphonia resulting from an involvement of the larynx. The neck, the chest and the entire thorax are finally affected and, to a less degree, the thighs and the abdomen.

Abnormal *pigmentations*, often occurring early, form an integral

part of the picture; they are sometimes diffuse and limited to the sclerotic portions, often much more extensive, macular, mottled or plexiform.

In a few rare cases, the production of subcutaneous calcareous concretions has been noted, appearing primarily and predominantly on the fingers (Thibierge and Weissenbach, 1911). [A unique case of cutaneous ossification in scleroderma has been recorded by Pollitzer, 1917.]

The course is slow, interrupted by remissions; death is the result of complications or of cachexia; it may occur suddenly.

Atypical cases have been noted, beginning with sclero-edema and accompanied by erythema. The association of sclero-edema in patches with sclerodactylia has also been observed. The diagnosis of the clinical form may present some difficulties on account of these transitions which, moreover, justify the classification of all sclerodermas in a single group.

The *differential diagnosis* from Raynaud's disease may be impossible at the onset; although sclerosis of the skin is not the initial and dominating feature of this condition, there occur true mixed or intermediate cases.

Lepra nervorum is characterized by anesthesia, muscular atrophy and thickening of the nerves.

In syringomyelia, there exists a dissociation of sensibility, but no true sclerosis. A confusion with progressive arthritis deformans could be due only to faulty observation; the stiffness and the deformity of the fingers in this case are not referable to the condition of the skin, which is thinned but remains normal. Association is, however, possible and has been demonstrated.

The frequent pigmentations of scleroderma should be kept in mind in order to avoid confusion with other melanodermas.

Partial Sclerodermas.—The sclerotic lesions may be circumscribed, either in *patches* with well defined contours, or in *bands* and in *rings*.

Scleroderma in Patches.—The patches of scleroderma, also known as *morphea*, differ from cicatrices by their spontaneous and primary appearance as well as by their course. They are distinguished from the macular atrophies by their sclerotic character.

Morphea begins as a more or less thickened and indurated, lilac or purplish spot, which slowly increases in size; at the end of a few weeks or months its center becomes whitened and indurated, often through confluence of originally isolated sclerotic points. According as the patch is level, infiltrated, or mammillated, a distinction is made between morpheas *alba plana*, *lardacea* and *tuberosa;* but the appearance may change in the course of development. I have described above—under *Morphæa guttata*, white-spot disease—the form in which the induration resembles parchment and is merely superficial.

The patches of morphea have a variable extent, from 1 to 20 cm., or larger; an oval or irregular shape, with convex or sinuous borders; of a glistening, pearly, bluish, or wax-white color. They are sometimes spotted with pigment, or mottled with telangiectases, or they may desquamate in large shreds. Their most characteristic feature is that they are usually bordered by a zone of mauve, purplish or tawny color, several millimeters in width, constituting the lilac ring of authors. The consistence of the patches is hard, even woody; they cannot be folded; sometimes they are more or less closely adherent to the subjacent layers, the bones and the muscles.

On these patches the hairs have fallen out, the secretions are absent, sensibility is lessened in proportion to the degree of the sclerosis; at the onset, tickling and itching sensations have been noted.

Morphea spots may be single or multiple, sometimes symmetrical and occupy any region of the body. They are not uncommon in the face. On the hairy scalp, confusion must be guarded against with the cicatrices from lupus erythematodes or other causes. On the chest, they must not be confused with scirrhus cancer. They are also seen on the abdomen, often on the limbs and even on the buccal mucosa; in the last named location, they assume the shape of hard white spots, which are retracted, in contradistinction to leukoplakia.

The patches of morphea after remaining stationary sometimes for a very long time, may begin to spread; or they may give rise to obstinate ulcers at points exposed to injury. Most commonly, they finally undergo retrogressive changes, the lilac ring disappears, the extent of the patch narrows, its center shrivels, becomes supple and covered with superficial telangiectasis. A localized atrophy is left behind, known as *morphea atrophica*. It is said that total disappearance may occur.

Scleroderma in Bands.—Instead of patches, the sclerosis may form bands, 2 to 5 cm. wide, with broadening and narrowing here and there. Their length is variable; they may extend, for instance, from the shoulder to the hand, or from the pelvis to the heel. The sclerotic band is prominent or level, or depressed as a groove, and may impede movements. The lilac ring is rarely complete.

A relatively not uncommon clinical type (numerous cases of which were presented at the London International Congress, 1896) is is that of *frontal scleroderma;* a white or brownish depressed sclerotic band, starting from around the superior orbital foramen, passes across the forehead, like a sabre-stroke, terminating more or less close to the fontanelle.

A possible relation of the course of sclerodermatic bands with the nervous, radicular, etc., territories has been pointed out; but these relations are irregular and very inconstant (see linear nevi, p. 206).

Annular Sclerodermas.—In exceptional cases, a band of scleroderma has been seen to hollow an annular or semiannular groove around a limb or around a finger. The stricture may produce edema and elephantiasis below it.

Analogous appearances may be produced by two other affections, positively distinct from annular scleroderma.

One of these is *ainhum*, of Da Silva Lima—or spontaneous amputation of the toes—which is endemic in several colored races and begins in adult life, almost invariably at the little toe, which it strangles and finally separates.

The other, known as *congenital amputations*, is observed in all races, affects the limbs at any level and is attributed to an intrauterine constriction by amniotic bands. Separation is incipient at birth and may become complete in a few weeks or years.

Pathological Anatomy.—The lesions of scleroderma, no matter of what clinical form, consist of a thickening with partial disappearance or sometimes a degeneration of the connective-tissue bundles; the elastic fiber plexus is preserved and appears increased as the result of close approximation of the fibers. It is not known if this fundamental lesion is the outcome of an always identical process.

In recent cases of generalized scleroderma and scleroderma in patches, I have demonstrated a subacute, predominantly perivascular inflammation and a newformation of connective tissue. The smooth muscles may be hypertrophied. The vessels were almost invariably found to be affected by endoperiarteritis and phlebosclerosis; the peripheral nerves are slightly involved, or their sheath is thickened.

The papillary body is flattened and obliterated, the epidermis is often atrophied; the horny layer is increased. The follicles and glands disappear. Sclerosis of the hypoderm and of the muscles has been described, with inflammation of the periosteum and rarefaction of the bony substance; incongruous lesions, of doubtful significance, are found in the nerve centers.

In a case of generalized scleroderma, of hyperacute onset, observed by Thibierge, I found the following changes: in the sclerematous stage, a thickened epidermis, a tendency to obliteration of the papillæ, a condensation of the papillary body where the elastic plexus was irregular and ravelled; in the stage of sclerotic atrophy, a thinned epidermis apparently glued to the corium, disappearance of the papillæ, a narrow and sclerotic papillary body supplied with several layers of elastic fibers parallel with the surface; no sign of a cellular infiltration.

Etiology and Pathogenesis.—Scleroderma is more common in the female sex. The diffuse forms are met with especially between twenty and forty years, the partial forms, at any age.

Sudden exposure to cold, violent emotions, overexertion, menstrual disturbances, are frequently held responsible; the probable action of traumatism has been pointed out for the localized and even for the generalized forms. It seems that several infections may play a part, especially acute rheumatism, typhoid fever, etc., perhaps tuberculosis or congenital syphilis. In a general way, very little is known concerning the etiology of the sclerodermas.

The *pathogenesis* is equally uncertain. Vascular lesions are practically sure to occur. The effect of a trophic or angioneurotic nervous disturbance has been suspected, on account of the arrangement of certain sclerodermas in bands and the common nervous antecedents of these patients. Cases where sclerodermas supervened in the course or as a sequel of exophthalmic goitre have given rise to the thyroid and polyglandular theory. Various infections or toxic influences possibly act upon the nervous system and the vascular apparatus, through the intermediation of functional disturbances of the internally secreting glands. [The probabilities seem to me to indicate that endocrinal disorders are the most likely etiological factor.]

Treatment.—Nearly every kind of internal medication has been tried in the diffuse sclerodermas; there are no specifics and each case will have to be treated according to its own indications.

Salicylates have seemed to be useful to me; others have advocated the iodides, arsenic and fibrolysin. Successful results have been obtained with thyroid treatment, cautiously and persistently administered, but are unfortunately not constant.

Aside from good hygiene, advantageous use may be made of hydrotherapy, the continuous current, electric baths, massage, treatment with sulphurous or chlorinated waters, various hot springs and mud-baths.

In the partial sclerodermas, negative-pole electrolysis may be recommended; the punctures must be applied at considerable intervals and only weak currents be used; ionization has likewise yielded very encouraging results. Massage, salicylic acid or salol ointments are sometimes beneficial. Mercurial plasters have a classical reputation. Several of these treatments may, moreover, be combined.

REGIONAL ATROPHIES AND DERMATOSCLEROSES.

In addition to the macular sclerodermas and atrophies, there exist a certain number of clinical types in which the lesions, while morphologically analogous, are distinctly *regional*.

Whether they be related or not to the affections described above,

the identity of the dermatological lesion leads me to consider them in this connection.

Facial Hemiatrophy.—This affection—also known as *trophoneurosis facialis* or *aplasia laminaris*—consists of a very marked thinning of the skin of one-half of the face, without sclerosis or adhesion. The atrophy extends to the corresponding half of the palate, the velum and sometimes the tongue. The prominences of the bony framework are likewise reduced. The affected side seems to be changed as if by old age and situated on a plane behind its normal. The skin is white or pigmented; the sensibility is intact, but anidrosis and alopecia are present.

This very rare affection begins in youth in the form of patches which gradually spread. Some authors compare it with scleroderma. Cases of coincidence of this disease with a scleroderma in extensive patches have been reported.

Kraurosis Vulvæ.—The term "kraurosis" (Breisky), the meaning of which was rather vague, has been more accurately defined especially by the work of Jayle, and must be reserved for a progressive sclerotic atrophy of the cutaneo-mucous tissues of the vulva, gradually leading to stenosis of the vaginal orifice, disappearance of the labia minora, the prepuce and frenum of the clitoris and the obliteration of the labia majora. [I have seen the process extent to the anal orifice.] The mucosa of the affected regions is always smooth, glistening, and dry; its color is white, or red, or mottled. Complication with leukoplakia is common and, in this case, cancer is not rare.

The exclusive or principal pathogenic condition of kraurosis seems to be the suppression of the ovarian functions through senile involution, sclerotic atrophy, or castration; syphilis seems to play a part in some cases.

Kraurosis must not be confused with simple vulvar leukoplakia nor with the white coloration of the vulva through lichenization, which occurs in prolonged cases of vulvar pruritus. Aside from treatment with warm irrigations and the high frequency current, extensive excisions must not be delayed in cases of threatened cancer.

Dermatoscleroses of the Legs.—In many adults and in old people the skin of the legs is the seat of very polymorphous but equivalent pathological changes which may terminate either in sclerotic atrophy or in elephantiastic pachyderma.

In the etiology, the patient's sex is of small importance; but predisposing conditions are represented by an age between thirty and forty-five years, laborious occupations requiring prolonged standing, repeated pregnancies, phlebitis, traumatisms, etc. Recently, the influence of syphilis and tuberculosis has been investigated. The essential factors seem to be arteriosclerosis and

especially varicosities. Both legs are usually involved, although in different degrees.

Varicosities, especially the deep and slightly apparent ones, lead to congestion and lowered nutrition; edema and hematic pigmentations follow; the soil is prepared for complications, varicose eczema, phlebitis and ulcers, which further aggravate the nutritional disturbance by giving rise to thromboses and opening an avenue to infections, lymphangitis, etc.

The resulting dermatosclerosis, which is alone to be discussed in this place, may be diffuse or circumscribed.

In the former case, the *diffuse* form, the skin is adherent to the tibia and the aponeurosis, of a pasteboard-like or woody hardness, impossible to raise or fold; its color is earthy or checkered with purple or brown, with depressed white spots. Its surface is smooth, shining or scaling and cracked; sometimes it is covered by a thick layer of dry or oily brownish crusts under which may be found pinkish, moist or plainly eczematous surfaces. These changes surround the entire circumference of the leg and extend as far up as the knee; the foot is usually merely edematous and the nails onychogryphotic.

In the *circumscribed* form, there exist one or several hard, pinkish or pigmented patches, level with the skin or slightly depressed, deeply adherent, often extending into the hypoderm in thick nodular strands. These are sclerotic foci of periphlebitic origin. A variety of reticulated sclerosis, of checkered hue, is also met with, especially in the neighborhood of the ankles.

This dermatosclerosis is differentiated from progressive scleroderma by its definite localization; from scleroderma in patches by the absence of a distinct boundary and a lilac ring; from the cicatrices of ulcers, which often exist at the same time, by the absence of a distinct thickened border, which is characteristic of the latter.

The efficacy of the *treatment* depends upon the degree and duration of the lesions. Complete rest in bed, with elevation of the legs, cleanliness and the dressings required by the condition of the skin, lead to progressive improvement, which is often considerable and may be further augmented by massage and radiotherapy. The patient must be instructed to wear roller-bandages or elastic stockings.

CUTANEOUS DYSTROPHIES.

Xeroderma Pigmentosum.—This dystrophy was first described by Kaposi and also bears the names of *melanosis lenticularis progressiva* (Pick), *atrophoderma pigmentosum* (R. Croker), and *epitheliomatosis pigmentosa* (E. Besnier). It is familial and of congenital origin, although it does not manifest itself until the first years of childhood,

The influence of consanguinity of the parents has been shown by statistics (11.8 per cent. of the cases). Sometimes the children of one sex are alone affected

The lesions of xeroderma pigmentosum, resulting from an abnormal sensibility to light-rays, are essentially regional and affect the uncovered regions, face, neck, hands, forearms, sometimes the legs and the feet, rarely the trunk.

At the onset, usually in the spring or summer and after a solar erythema, the skin is seen to become covered with lenticular pigmentary spots of the appearance and size of freckles or larger. It soon becomes abnormally dry and peels in fine lamellæ, and later is mottled with telangiectases and atrophic white spots. The latter

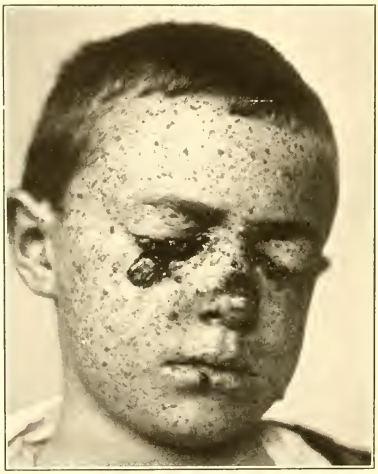


Fig. 113.—Xeroderma pigmentosum. (Author's photograph.)

may follow upon impetiginous lesions or verrucosities, or originate spontaneously. Finally the tissues become atrophied and contracted, whence ectropion, atresia of the mouth, thinning of the nose and ears, etc.; sometimes conjunctivitis and photophobia are noted.

The mottled appearance resulting from atrophic and red spots, stellate telangiectases and pigmentary spots, is characteristic.

More or less delayed, often about the age of eight or ten years, various newformations appear upon this background (Fig. 113); dry verrucous elevations, red and flabby protuberances of sarco-

matous or angiomatous appearance; finally, epitheliomas of various types, fungoid or ulcerative, which usually result from the malignant transformation of the warty growths. These various tumors may sometimes heal; but, as a rule, the epitheliomas become mutilating, involve the glands and lead to early death, usually before the age of twelve years. A few individuals, however, have lived to the age of forty.

The *pathological anatomy* affords scanty information as to the nature of the disease. Epidermic hypertrophy and atrophy of the derma are demonstrable; the various neoplasms mentioned have their ordinary structure, the epitheliomas are sometimes tubular, sometimes lobulated.

The *nature* of xeroderma consists primarily of a cutaneous malformation of the type of nevic diseases, characterized by hypersensibility of the skin to light radiations which leads secondarily to a degeneration analogous to the degenerative changes of presenile and senile dystrophy, radiodermatitis and arsenic poisoning.

The *prognosis* is very grave, but varies with the severity of the case and the treatment.

The *treatment* consists principally in the avoidance of sunlight; the covering of the skin with protective pastes containing quinine salts or aesculin; sometimes, it is useful to cover it with masks of mercurial or red oxide plaster.

Special care must be taken to destroy one by one, and very promptly, the neoplasms which develop, even the simple verrucosities, making use of the curette, the bistoury, the galvanocautery, caustic agents, or electrolysis; radium has recently been recommended. Internal medication is apparently of no use.

Senile Degeneration.—The degeneration or senile atrophy of the skin, which regularly occurs at an advanced age, begins sooner or later after the age of forty, according to the mode of life, the general health and the heredity of the individual.

Exposure to inclemencies of the weather plays a certain part, the uncovered portions, like the face, the neck and the back of the hands and wrists, being the earliest and worst affected; bad general and local hygiene, excesses, loss of sleep and diseases of all kinds, also act as predisposing factors.

Senile degeneration manifests itself as changes in the thickness and color of the skin, dryness and a diminished plasticity, which results in wrinkles.

Two types may be distinguished and are often associated:

The most common is *simple atrophy*, characterized by a parchment-like thinning, a yellowish, grayish or reddish color and transparency of the skin, rendering visible the veins, muscles, and tendons, etc.; its surface is shining or in a state of ichthyosiform

xeroderma. Pigmentary or achromic spots, telangiectasis and sometimes purpura senilis are often present at the same time. Aside from the above mentioned regions, the extensor surfaces of the joints are the most affected.

The second type is *colloid atrophy,* in which the skin is not thinned, sometimes on the contrary thickened, but of a straw-yellow or old ivory color, an uneven surface like orange-peel, soft and flabby, distended and folded. This condition is observed especially on the neck and the entire face, except the cartilaginous region of the nose.

Histology shows as the principal lesion an alteration of the elastic fibers. In the atrophic type, they have only become basophilic (elacine of Unna) and the connective tissue is rarefied. In the colloid type, they are furthermore swollen and perhaps combined with the substance of the connective-tissue bundles (*collastine* and *collacine*). In this case there is found under the epidermis a continuous felt-like band, from $\frac{1}{3}$ to $\frac{1}{2}$ mm. in width, staining black with acid orceine; this is the *diffuse elastoma* of Dubreuilh (1913). Several authors have erroneously confused this relatively commonplace lesion with pseudoxanthoma elasticum. The epidermis is thinned and hyperpigmented. The papillæ are short; the blood-vessels are dilated and surrounded by cells. The glands are atrophied.

Both types of senile degeneration predispose to the senile keratoses which may lead to multiple epitheliomatosis.

The *treatment* must be prophylactic and consists in correct hygiene. Facial massage, which is wrongfully credited with the virtue of curing wrinkles, may seem useful for a while, but is followed as a rule by an exaggeration of the lesions.

Presenile Dystrophy.—On the uncovered portions of the skin, in persons exposed to all kinds of weather, sailors, automobilists, aviators, coachmen, farmers, mountaineers, etc., changes entirely analogous to those of xeroderma and even more so to those of senile degeneration may be observed after the age of twenty-five or thirty years.

Diffuse atrophy, pigmentations, cyanosis, telangiectasis and keratosis are met with; this dystrophy also terminates in multiple epitheliomatosis.

All authors point out the striking resemblance often presented by the skin affected with chronic *radiodermatitis* to that of xeroderma and the senile and presenile dystrophies. The analogy extends to the identity of the epitheliomatous complications which are here equally frequent.

Pseudoxanthoma Elasticum.—Under this name I designated, in 1896, a rare affection, characterized clinically by a yellow hue mixed with lilac, with thickening, softness and relaxation of the skin in certain regions. It appears in youthful individuals and in adults.

The changes are preferably localized in the vicinity of the great articular folds, groins, axillæ, bends of the elbow and even on the neck; it has never been observed in the face. Furthermore, around the dystrophic surfaces are seen perifollicular spots of the same appearance, of slightly prominent, soft, yellowish papules.

However, the analogy to xanthoma is only apparent. Histologically the condition is a degeneration of the elastic network of the deep portions of the corium; its fibers swell, proliferate, split and break up into fragments. I have given to this special lesion the name of *elastorrhexis*.

Certain authors, especially impressed by the elastic hyperplasia, have interpreted it as an *elastoma* or *hamartoma*, regarding this degeneration as a sort of widespread tumor.

At any rate, the nature of the lesions of pseudoxanthoma (elastorrhexis), their seat and distribution, as well as the age at which the affection appears, plainly distinguish it from the ordinary senile colloid degeneration, or diffuse elastoma. It progresses slowly and persists indefinitely.

Colloid Milium.—Also named *miliary colloid degeneration of the cutis;* this rare dystrophy first described by E. Wagner, then by E. Besnier, manifests itself as translucid, soft yellowish elevations, disseminated or conglomerated, situated on the face, the neck and the upper limbs. This condition should accordingly be studied rather with the benign tumors of the skin (XXXI).

CHAPTER XVIII.

CUTANEOUS HYPERTROPHIES.

Under the name of cutaneous hypertrophy or *pachyderma*, I designate a persistent increase in thickness of the skin as a whole, due to an interstitial fibrous hyperplasia.

Partial thickenings, affecting the epidermis alone, or the papillary body, have been discussed elsewhere (Chapter XI and XII).

Fibrous or adipose hypertrophy of the hypoderm alone, without thickening of the derma does not belong to the domain of dermatology.

Cutaneous hypertrophy is only very rarely generalized, but may be very extensive; it is usually regional. The boundaries of the change are almost invariably rather indistinct.

In pachyderma, the skin is thickened to a variable degree and changed in its consistence. As a rule, it is firm, unyielding, or actually woody; it is not at all or very slightly depressible, and the indenting finger leaves no dimple; it is incompletely reducible by compression *en masse;* it adheres to the subjacent tissues and is not easily raised in a fold. Sometimes, however, its consistence is softer and more elastic. The condition of the surface and the color of the affected regions vary greatly in different cases.

Cutaneous hypertrophy must be distinguished from three related processes, which may, moreover, be combined with it in variable degrees:

Inflammatory infiltration is due to deposits of embryonic cells, or cells derived from the blood, in the tissues; it behaves like the acute inflammations or, when subacute, it is as a rule more or less circumscribed and follows a progressive or a retrogressive course. In both cases, however, it sometimes terminates in hypertrophy.

Edema, the result of a fluid exudate which infiltrates the tissue, is depressible, plastic, retains the imprint of the finger and is entirely reducible by compression *en masse*, even in chronic cases. Edemas of mechanical or dyscratic origin, such as those of cardiac or Bright's disease, etc., never lead to pachyderma. Inflammatory edemas, on the contrary, are not infrequently the starting-point of pachydermas, with which they are connected by imperceptible transitions, as illustrated by the often used term of *elephantiastic edema*.

Tumors are circumscribed, heterotopic or hyperplastic new-growths instead of simple hypertrophies. The question often arises,

however, in which group a given swelling properly belongs; for instance, an elephantiasis limited to an eyelid or a labium majus may be mistaken for a myxoma, or a bunch of lymphatic varicosities may have the appearance of a lymphangioma, etc.

Hypertrophic Dermatoses.—The typical form of pachyderma, whose clinical features and lesions conform to the definition given above, is called *Elephantiasis arabum* (in contradistinction to *Elephantiasis græcorum*, which is leprosy), or briefly *elephantiasis*. It will be discussed first in order.

Another section will comprise a series of affections which I group under the heading of *non-elephantiastic hypertrophies*. These form a very heterogeneous group. The differential diagnosis of the genuine elephantiasis will be discussed in dealing with them.

ELEPHANTIASIS.

Elephantiasis is a regional cutaneous hypertrophy characterized by its course and its pathogenesis.

According to the etiological factors, the following clinical forms are distinguished: *Elephantiasis nostras; secondary elephantiasis; filarial elephantiasis and congenital elephantiasis.*

The symptomatology of elephantiasis in general, irrespective of its origin, is as follows: The elephantiastic process begins with *edema*, the inflammatory character of which is sometimes clearly evident, sometimes not noticeable. Ultimately, a firm non-plastic tumefaction is established, constituting sclerotic or elephantiastic edema. In the fully developed stage, *pachyderma* is present, combined with a variable degree of hypertrophy of the subjacent tissues and associated with all the attributes mentioned above. This process is apparently brought about through the association of three pathogenic factors: lymphatic congestion, inflammation and venous congestion.

The course of the disease is intermittent; it is indefinitely progressive and more or less extensive.

Symptoms.—The affected regions are swollen, tense and hypertrophied; their normal elevations and depressions are obliterated; sometimes, they are crossed by deep grooves.

The lower limb, for example, which on account of less favorable circulatory conditions, is the chief seat of election in elephantiasis assumes the appearance of a column or an elephant's leg (Fig. 114). The integument is enormously thickened and adherent to the deep tissues. Its consistence, which is that of a doughy firmness at the thigh, usually becomes harder and more resistant as it approaches the malleoli where it is sometimes woody.

The surface may be smooth and of normal color, or purplish, or

brownish; again, it may be covered with laminated scales, or cracked hyperkeratoses; in the majority of the cases, it is covered by more or less conglomerated verrucosities, of unequal size, rounded and of the size of a millet-seed to that of a cherry-pit; or papillomatous, acuminate or obtuse and crowded together. These verrucosities may be pinkish or white, slightly reducible and translucid like

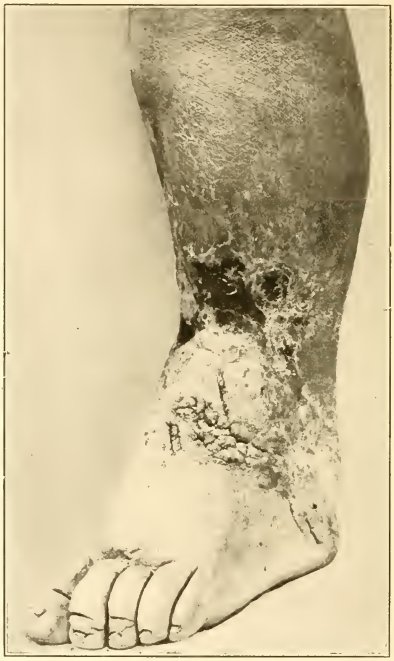


Fig. 114.—Elephantiasis nostras, consecutive to an ulcer of the leg.

large vesicles; in this case, they are due to lymphangiectases or lymphatic varicosities, which can be pricked with a needle and yield a profuse, prolonged flow of lymph (*lymphorrhea*); in other cases, they are hard, polygonal through mutual pressure and often covered with a hyperkeratotic layer, of dry or oily consistence, of a dirty gray or blackish color. Under the crusts, as well as in the grooves, a macerated and fetid epidermis is exposed and sometimes ulcers

are present having an irregular outline and floor with a sanious discharge.

On the legs, elephantiasis is unilateral or bilateral. When it is caused by a local lesion, such as an ulcer of the leg, the pachyderma develops below this lesion, consequently on the foot. When the causative lesions are situated higher up, the elephantiasis is on the contrary often limited by the malleoli, forming an enormous cushion which is separated from the foot by one or several deep grooves. The foot may retain its normal volume, but is usually swollen and verrucous in its dorsal region, chiefly near the toes and above the heel.

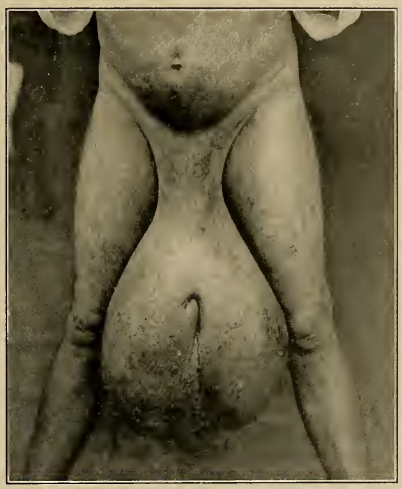


FIG. 115.—Elephantiasis of the scrotum, of filarial origin in an Arab. Case and photograph of Dr. Raynaud, Algiers.

The thighs are invaded from below upward, or consecutively to elephantiasis of the external genital organs.

The latter constitute the second seat of election of elephantiasis. In men, hypertrophy of the sheath of the penis may transform this organ into a pear-shaped mass 20 to 40 cm. in length. When the scrotum is the seat of elephantiasis, it may become as large as or larger than an adult's head (Fig. 115), engulfing the penis; its surface is smooth or verrucose. The name *lymph-scrotum* is applied to elephantiasis of the scrotum with very pronounced lymphatic varicosities.

In women, various portions of the vulva, especially the labia majora and minora, or only one of these, assume an enormous size; the condition may simulate a myxoma (Fig. 116). I have elsewhere mentioned the syndrome of esthiomene, which may be brought about by a variety of vulvar ulcerations accompanied by elephantiastic hypertrophy.

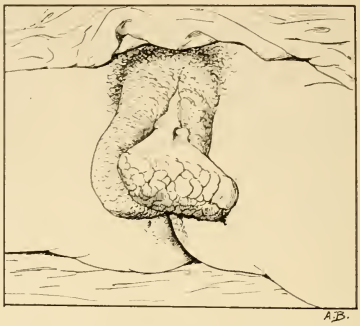


Fig. 116.—Elephantiasis nostras of the vulva.

In the groins, elephantiasis gives rise to general tumefaction with enormous lymphatic dilatations, known as *adeno-lymphocele.*

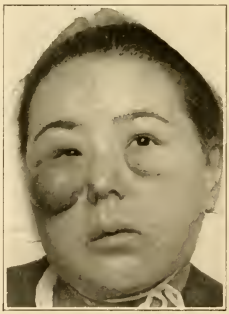


Fig. 117.—Elephantiasis of the face; elephantiastic edema of the eyelids, following repeated attacks of erysipelas.

The upper extremities which are rarely attacked separately, are transformed into monstrous sausages, constricted at the elbows and the wrists.

In the face, elephantiasis generally appears in the form of permanent bloating, a flabby but non-plastic edema, usually consecutive to recurrent erysipelas. It may occupy the entire face and be accompanied by lymphatic varicosities of the mouth. In other cases it predominates in a given region, for example at the ears. The eyelids, especially the lower lid, may become the seat of a smooth pseudo-myxomatous globular swelling (Fig. 117). On the lips, nose and chin, elephantiasis is usually secondary to the lesions of lupus or leprosy, or especially sclero-gummous syphilides; the resulting facies is called *leontiasis*.

It is fairly common, in all kinds of elephantiasis, for several territories to be invaded at once or consecutively, for instance a lower limb and the genital organs or a leg and an arm, etc.

At the borders of the elephantiastic regions, the transition into the normal condition is always gradual; the intermediate zone is edematous and soft.

The corresponding glands are always altered, usually indurated and involved in the general swelling; they may be impalpable in the fibro-edematous swelling.

Pathological Anatomy.—The elephantiastic tissues are tough, resistant under the knife or merely firm, but always translucid, of gelatinous appearance, gorged with plasma which escapes abundantly from the cut surface. The cutis, the thickness of which may reach 2 or 3 cm., the hypoderm two or three times thicker, with the muscles and aponeuroses, etc., form a single lardaceous mass which extends as far as the bones which also are sometimes hyperplastic. The vessels, especially the veins and the lymphatics, are seen gaping in cross-sections, producing a riddled or cavernous appearance.

The microscope shows in all cases a new formation of young or of fibrous connective tissue, without an elastic plexus, sometimes purely interstitial, sometimes forming besides an additional layer between the corium and the greatly hypertrophied papillæ. Between the connective-tissue bundles, the connective cells are hyperplastic, sometimes of giant size; collections of leukocytes and plasmocytes are also met with. The vascular walls are thickened, infiltrated with cells, or sclerotic. The muscles and glands are atrophied. The adipose tissue often seems to be increased.

Briefly, the inflammatory character of the lesions and on the other hand the venous and lymphatic stasis, are distinctly evident; all the supporting tissues are hyperplastic.

The corresponding *glands* are sclerotic, as a rule; sometimes they are found to be transformed into a fibrous shell, invaded by fatty degeneration or into lymphatic cavernous tissue. In secondary elephantiasis, they are often degenerated. In an operation upon

tropical elephantiasis of the scrotum, an adult filaria was found in the excised tissues.

I have repeatedly observed remains of *phlebitis obliterans* in the large veins supplying the elephantiastic regions.

Pathogenesis.—The most plausible explanation of the lesions of elephantiasis refers them in the first place to stasis, especially lymphatic but also venous, leading to edema; and to a consecutive, or rather concomitant, irritation of the connective tissues. Cruveilhier particularly stresses the venous lesions, Virchow, the glandular obstruction.

Although experimental elephantiasis cannot be arbitrarily produced, it has several times been involuntarily caused in man through the extirpation of a group of suppurating or sclerotic glands, or through paraffin injections which have obliterated a lymphatic plexus.

Various infections or neoplastic processes may result in the conditions essential to the development of elephantiasis; local tuberculosis, tertiary syphilis and cancer may become complicated by secondary elephantiasis.

The relation of filariasis to the endemic elephantiasis of tropical countries is no longer questioned, but superadded infections may be suspected of playing a certain part in such cases.

The relations of elephantiasis nostras to ordinary lymphangitis had long been known when Achalme and Sabouraud demonstrated the presence of the *streptococcus* in the elephantiastic tissues in the course of acute attacks. Later it was shown that other microorganisms may take its place.

The infection is exogenic and primary, or it may be secondary, for instance to ulcerative lesions, in other cases it seems to be endogenic, derived from remote foci, or lurking for a long time, even indefinitely, in a state of latent infection in territories which have once been invaded. Those who are familiar with the lymphangitic, phlebitic and glandular lesions of streptococcus and of erysipelas in particular, will not be surprised to see that elephantiasis may result therefrom.

Treatment.—An acute attack should be treated, like all cases of lymphangitis, by absolute rest, with elevation of the affected limb and moist dressings, or applications of ichthyol, thiol, etc.

Elephantiasis in the sluggish state must first be cleansed from the crusts and coatings by means of baths, fomentations, and oily inunctions; ulcers should be disinfected and dressed. Next, always assisted by rest in the position most favorable to decongestion of the affected part, systematic massage should be employed with compression by cotton-padded bandages, or preferably elastic bandages, proceeding cautiously and closely watching developments. The

results obtained by these methods are sometimes remarkable, but nevertheless as a rule incomplete. Castellani adds to the elastic compression, which must be kept up night and day for three to six months, daily injections of 2 to 4 cm. of fibrolysin. In case of syphilis, specific treatment would of course be indicated. Anti-streptococcus serum seems to be of slight efficiency. In hard elephantiastic edemas, Denis recommends injections of "du Breuil" water.

When sclerosis predominates and compression is no longer successful, galvanic electricity, advocated by Moncorvo and Silva da Araujo, is very valuable for softening the indurated tissues. The negative pole is represented by a bath in which the affected part is placed, or by moist compresses in which it is wrapped, the positive pole being applied to the healthy tissues; strong currents are required. Still better results can undoubtedly be accomplished by means of ionic medication. Attempts to perform "lymphangioplasty," through subcutaneous filiform drainage, are very variably estimated.

Some cases of elephantiasis, notably of the scrotum and penis in hot countries, constitute a disease against which extensive surgical ablations are the only efficient measure. In case of filariasis, injections of arsenobenzol will cause the filariæ to disappear from the blood, but they do not seem to act upon the elephantiasis.

Elephantiasis Nostras.—This is observed in adults of all ages, and in both sexes.

In a considerable number of cases, the portal of entrance of the infection, usually streptococcic, is obvious—a scratch, an incised corn, or any kind of erosion. The first lymphangitis may be of violent, abrupt onset, with edematous and painful progressive redness, long red streaks, glandular swelling and associated febrile malaise. Sometimes, especially in the face, it appears as a typical erysipelas. All the symptoms subside after a few days, but there follow repeated, sometimes rhythmical recrudescences, following traumatism, fatigue, exposure to cold, or ordinary causes; these are often less violent but more protracted than the first attack. The tumefaction, which had not entirely subsided before, becomes established and gradually increases.

In other cases, the initial lesion remains unknown. The attacks are afebrile and are marked only by heaviness or tenderness of the affected region, articular pains, etc. The lymphangitis in this case is not apparent, but nevertheless everything points to the occurrence of a lymphatic obstruction.

Such an obstruction appears evident in a third group where the elephantiasis develops as the result of an adenopathy. Tuberculosis of a group of glands, cancerous adenopathy, sclerotic adenopathy

following upon syphilis, a suppurating glandular inflammation, or surgical extirpation of glands, may lead to elephantiasis of the corresponding territories. One of the lower limbs, or the genital organs, or both these regions together, will be involved in the case of inguinal adenopathy (Fig. 118); the upper limb, as a sequel to cancerous axillary adenopathy, which is so common in the course of cancer of the breast; the face, in the case of submaxillary adenopathy.

Fig. 118. — Elephantiasis nostras of the lower limb with lymphatic varicosities of the scrotum and at three points on the thigh. The lesions in this young man were consecutive to tubercular adenopathy of the inguinal glands. Thèse de Guillemin, Paris, 1900.

Gradually, without inflammatory attacks, the above outlined clinical picture develops; lymphatic varicosities, lymphoceles and lymphatic fistulas are especially frequent and important in this group of cases.

Secondary Elephantiasis or Elephantiastic Conditions.—This is by far the most common form of elephantiasis in our climates. In these cases, sclerotic edema, then elephantiastic, usually verrucous pachyderma develop in connection with a local lesion of which they represent a complication.

Elephantiastic conditions are observed in the course of cutaneous tuberculosis and especially lupus of the limbs (elephantiastic lupus); in tertiary syphilis, especially as the result of recurrent gummous infiltration on the limbs or on the genital organs, also at the circumference of the mouth and nose (syphilitic leontiasis); in leprosy, when incurable ulcers are present; in the course of leg ulcers, where one may observe the formation not only of a callous cushion, but also of monstrous deformities (Fig. 114), above the ulcer or its cicatrix.

The pathogenesis of these elephantiastic conditions is often complicated, so that it would not be justifiable to divide them into tuberculous, syphilitic, etc., elephantiasis. The specific process undoubtedly invades the lymphatics and veins of the affected region; but in addition, the corresponding glands are degenerated or sclerotic; finally, secondary infection of the ulcerations by the streptococcus or other microbes, whether revealed or not through attacks of lymphangitis, is extremely probable and can be bacteriologically demonstrated in some of the cases.

It is useful to know that after syphilitic chancrous lymphangitis, it is not uncommon to observe a sclerotic edema of the prepuce and sheath, or of a labium majus, undoubtedly due to a specific lymphangitis through the spirochete; this lesion may subside more or less gradually under the influence of antisyphilitic treatment.

Elephantiasis Filariosa.—In many tropical countries, cases of elephantiasis are extremely common; some are probably referable to the various pathogenic factors mentioned above, but in addition there occurs an endemic helminthiasis, known as *filariasis*, which may give rise to lymphangitis, orchitis, lymphangiectasis, lymph scrotum, filarial abscesses, chyluria, chylous hydrocele and also frequently to elephantiasis.

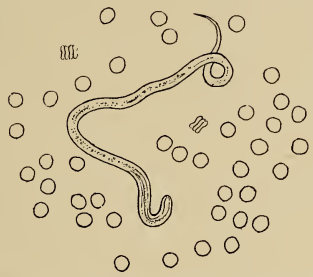


FIG. 119.—Filaria sanguinis hominis, surrounded by red corpuscles. Drawn from a fresh specimen prepared by Dr. Jolly. × 300.

It is caused by the *filaria Bancrofti* and its embryos, the so-called microfilaria. This worm is a nematode, the adult female, discovered by Bancroft in 1876, measuring 8 to 10 cm. in length, while the male is considerably smaller; they lodge, a few in number, in the large lymph trunks or glands, where the female hatches innumerable embryos which resemble small active eels, about 300μ long, from 7 to 9μ wide, enclosed in a glassy sheath. These microfilarias were seen by Demarquay (1863) in a chylocele and by Lewis (1877) in the circulating blood (Filaria sanguinis hominis) where they are found only during the night and should be looked for about midnight in doubtful cases; in the daytime they withdraw into the pulmonary vessels, according to the observation of P. Manson.

The disease is transmitted through mosquitoes, which in stinging the patients absorb the embryos; these undergo a metamorphosis in the thoracic muscles of the insect and are then inoculated into other human subjects, where they reach maturity.

The etiological role of these parasites in elephantiasis seems to rest on the very frequent association in the same countries and

in the same patients, of monstrous pachydermas and other symptoms of filariasis. It must be admitted, however, that numerous filaria-bearers have no elephantiasis and that on the other hand in many elephantiasis cases in hot countries, the embryos cannot be demonstrated in the blood.

It is accordingly possible that the obstruction of the large lymphatic trunks by the adult filaria, or of a large number of lymph vessels by ova which have not reached maturity (P. Manson), or the invasion of the embryos into the connective-tissue interstices of the affected parts, are sufficient causes of the elephantiasis; it is also possible that the filariasis constitutes merely a predisposing cause of ordinary elephantiasis.

At any rate, filarial elephantiasis is observed in infected countries, in all races, in both sexes, at all ages, but especially in adults. In 95 per cent. of the cases it occupies the lower limbs, one or both; very frequently also the external genital organs. It proceeds by lymphangitic or erysipeloid attacks, often with fever (filarial fever), with general symptoms and adenites, precisely as in elephantiasis nostras. In one case, Le Dantec was able to isolate and cultivate a "dermatococcus."

The symptomatic shades which distinguish tropical elephantiasis from the domestic form consist of its often excessive development and its frequent association with enormous lymphatic varicosities or other filarial manifestations.

Congenital Elephantiasis.—Under this term are united a long array of heterogeneous cases in which an enormous hypertrophy of a portion of the body was demonstrated at birth or shortly afterward. There is reason to suspect the following conditions, in different cases:

Giant lipomatosis; diffuse angiomas and lymphangiomas; lymphatic edemas due to tumors or malformations; and finally, neurofibromas or diffuse fibromatosis.

According to Moncorvo, who has made a special study of this subject, there exists a genuine fetal elephantiasis, due to intrauterine lymphangitis, the pathogenic agent having penetrated by the placentar route. Filaria have never been found in the affected children.

The localization is arbitrary; the hypertrophy affects a limb or part of a limb, the eyelids, the tongue, etc.

Congenital *macroglossia*, for instance, which is one of the most peculiar forms, has been referred to a variety of conditions in different cases, fibrinous or muscular hypertrophy, cavernous angioma and especially diffuse lymphangioma.

The subject is complex and as yet imperfectly understood.

NON-ELEPHANTIASTIC HYPERTROPHIES.

Not all regional or diffuse hypertrophies should be described as elephantiasis. As has just been seen, the diagnosis may present serious difficulties in the newborn; this is not generally the case in adults, except in rare cases.

By means of careful and systematic palpation, it may be recognized that there exists no true pachyderma in edema, in obesity, in the case of lipomas even when regional and symmetrical, in adenolipomatosis, in adiposis dolorosa of Dercum, in acromegaly, etc., which accordingly do not belong to the scope of this book.

More closely related to elephantiasis, although they remain distinct by their clinical and anatomical features and by their pathogenesis, are the following diseased conditions:

Neuro-arthritic Pseudo-elephantiasis.—The observations to which this name, due to Mathieu, is applied, as well as that of *trophedema of Meige* (1898) are difficult to interpret.

Extensive regional edemas, which become chronic and fibrous, occur in the absence of a demonstrable cause; in other cases, hard, non-depressible swellings are present from the start. The seat of predilection of this affection is on the lower limbs, only one of which may be involved, or both may be symmetrically swollen and enlarged; the genitals escape, as well as the foot, except sometimes its dorsal region. The trophedema may likewise affect the upper limbs and even the face.

At first sight the appearance is that of elephantiasis; but the skin always remains smooth, of normal color, without verrucosities or lymphatic varices; but it is adherent, and cannot be folded or indented.

The onset is often marked by severe neuralgic pains, or by spasms, without inflammatory symptoms; exaggerated tendon reflexes were noted by me in two cases. All evidences of a disease of the nervous system is absent as a rule.

The affection progresses or remains stationary, always lasting many years without other disturbance than an impairment of mobility; it may improve under the influence of thyroid treatment, massage and compression, or sometimes actually disappear.

The pathological lesions are not well understood. It is not known if this disease is comparable to myxedema, constituting an incomplete or abortive form, or to the diffuse hypertrophies which sometimes accompany infantile paralysis or paraplegia. It seems to be related to the following type:

Neuro-arthritic Pseudolipoma.—Potain, Bucquoy, Mathieu and others, have described under this name tumefactions having a supraclavicular and symmetrical seat of election, supposed to be

angioneurotic edemas, plastic at the onset, then fibrolipomatous, indistinctly outlined. Their true character has not yet been established.

Dermatolyses.—These malformations are always partial, usually congenital, but sometimes developing a considerable time after birth. They consist of thickening and well marked loosening of the skin in certain regions of the body.

Alibert recognized palpebral, facial, cervical, ventral and genital dermatolyses; still others may be observed, notably on the lower limbs. The denominations *pachydermatocele* (Mott) and *chalazoderma* (Bazin) are less commonly employed.

The relaxed skin forms large, thick and flabby folds which are dragged by their own weight so as to cover the subjacent parts, sometimes hanging down like an apron.

The majority of the cases of this group are probably referable to v. Recklinghausen's disease, in which the "major tumor" is represented by the pachydermocele. Swollen and nodular nerve-strands have been demonstrated therein (plexiform neuroma) and in several instances the patients were at the same time the bearers of smaller mollusca.

Ordinary relaxation of the skin due to pregnancy, senility, etc., is not true dermatolysis.

Cutis Hyperelastica, or Cutis Laxa, is a different malformation in which the skin of certain regions, without being distended or loosened, is of a doughy consistence, extraordinarily distensible, and suddenly resumes its place when liberated after stretching. Some of the "rubber men," exhibited in side-shows, can pull the skin of the neck up to the forehead, etc.

Myxedema.—Myxedema, or *pachydermic cachexia,* is a general dystrophy dependent upon thyroid insufficiency.

It is observed under a great variety of conditions, especially in women (Gull, Ord), or as a sequel of total extirpation of a goitre (*cachexia strumipriva,* J. Reverdin of Geneva and Kocher of Bern), also in children (*myxodematous idiocy* of Bourneville); the latter, which in combination with goitre constitutes *cretinism,* is known to be endemic in certain mountainous regions.

The principal *symptoms* are of a general kind: mental sluggishness, slowness of movements and of speech, anorexia, a retarded pulse, a lowered temperature and a sensation of chilliness; in children, idiocy and an enormous retardation in growth. Instead of dwelling upon these, I shall pass on to the external symptoms:

The integument is swollen, waxy white, dry, scaly, indurated and does not retain the imprint of the finger. The hairs fall out, the sebaceous and sweat secretions are suppressed.

The appearance is characteristic: the bloated "full-moon" face,

the hanging cheeks, the large nose, the swollen and open lips, convey an impression of imbecility. The buccal mucosa may be swollen and waxy. The neck is enlarged and the thyroid gland is not demonstrable on palpation. An analogous condition exists on the trunk and especially on the extremities, which may present a pseudo-elephantiastic appearance.

The hypothyroidism of the menopause, which is very common, gives rise to a greatly attenuated picture of myxedema.

The anatomical *lesions*, aside from atrophy, degeneration or sclerosis of the thyroid gland, in the *integument* consist of fibrous proliferation, with hypertrophy of the adipose layer.

I do not believe that "an infiltration of the tissues by a substance analogous to mucin" has been observed since Ord. But the following have been demonstrated: Collections of inflammatory cells (Virchow), which I was enabled to find also in experimental myxedema in animals; rarefaction with degeneration of the connective-tissue bundles; and a degeneration of the elastic fibers as in senile change.

The *treatment* with thyroid substance is imperative and yields remarkable results, but must be kept up indefinitely. Thyroid grafting has recently been tried.

Rhinoscleroma.—This is a progressive hypertrophic affection, described by Hebra and Kaposi, restricted to the region of the nose and upper lip; it is endemic in certain countries, such as Galicia, Hungary, southwestern Russia, etc., and is of microbic origin. [It is rare in America and almost always of exotic origin.]

The lesions usually begin on the septum of the nasal fossæ, or at the columella and the upper lip in the form of solid elevations, with a smooth and tense epidermis, of a red, pink, or pale color, which develop into a tumor of cartilaginous hardness resembling a keloid.

The disease affects both nasal fossæ, which become obstructed; the velum of the palate, causing retraction of the uvula; the pharynx and larynx, resulting in stenosis; and even the trachea.

The course is very slow and may lead after twenty years or more to death from pulmonary complications. The disease attacks youthful or adult individuals, especially of the indigent classes.

An encapsulated bacillus, the Frisch bacillus, closely related to, if not identical with the pneumobacillus of Friedländer, is held to be responsible and, it is claimed, has been found in pure culture in the glands, which according to Rona are frequently swollen.

The lesions consist of a sclerogenic cellular infiltration, containing large hyaline cells, the origin of which is referred by some to a cellular degeneration and by others to a degeneration of the bacilli themselves.

The surgical *treatment* is usually followed by recurrence; caustic

topical applications and interstitial injections are unreliable procedures. Radiotherapy is indicated and has furnished encouraging results. [I have definitely cured one case by means of *x*-ray. Autogenous vaccines should be tried in cases not amenable to radiotherapy.]

Rhinophyma and Hypertrophic Acne.—The nose is sometimes the seat of a pseudo-elephantiastic hypertrophy, giving rise to special clinical conditions in this region.

Although without actual gravity and hardly even inconvenient, it is readily understood that deformities of this kind are extremely distressing to the patients.

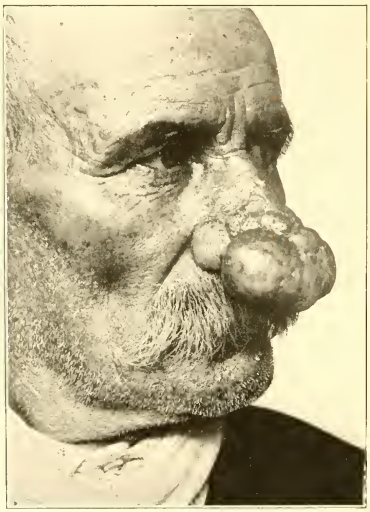


FIG. 120.—Acne hypertrophica of the nose.

These hypertrophies of the nose, more common in men than in women, do not occur until about the fiftieth year and affect only very *kerotic* individuals, who have suffered from acne when young; often they are complications of so-called "copper-nose." The course is progressive and very slow.

Two forms can be distinguished and are sometimes associated:

In the glandular variety—or hypertrophic acne of Vidal and Leloir—the skin is thickened, but of a normal color; the sebaceous pores are dilated and funnel-shaped; the point of a probe may be

introduced and a large amount of a sebaceous, vermicular and fetid material squeezed out.

In the fibrous angiectatic variety—or rhinophyma—the cutaneous surface is of a purplish red color, lumpy, furrowed by large-sized varicose venules, riddled with sebaceous orifices and often dotted with pustules.

In both forms, the nose is either uniformly increased in size, or covered with globular protuberances (Fig. 120) or even with pedunculated tumors attached to the lobule or the nostrils, sometimes reaching or exceeding the size of an egg and hanging down to the chin. Their consistence is flabby, uneven, gelatinous. The cheeks are sometimes invaded by an analogous proliferation. The boundaries of these changes are not well marked.

The lesions of hypertrophic acne consist of an enormous hypertrophy of the sebaceous glands, with an ampullary dilatation of their excretory duct. In rhinophyma, on the contrary, hyperplasia of the connective tissue and of the vascular and lymphatic plexus predominate. Disseminated foci of cellular infiltration are regularly present. Complication with epithelioma has been repeatedly noted.

The *treatment* demanded on account of the disfiguring character of these affections may consist at the onset of ichthyol applications and warm astringent lotions, or strong sulphur lotions, which in combination with massage and expression of the sebaceous material, may improve the condition of the affected parts.

It is preferable to interfere actively, without loss of time, by means of the galvanocautery, by scarifications, or electrolysis.

In the presence of a considerable hypertrophy, the operation known under the name of *decortication*, performed with the thermocautery or the bistoury, yields excellent results; grafts are unnecessary, for the remnants of the divided glands become centers of epidermic renewal; it is essential not to excise too deeply. Recurrences have not been recorded.

CHAPTER XIX.

FOLLICULOSES.

The forms of dermatological lesions which still remain to be studied in the first portion of this book have the peculiar feature of being characterized, not by the nature of the pathological process, but by its localization.

As folliculoses I designate those pathological processes which affect exclusively, or with evident predilection, the pilo-sebaceous follicles.

The syndromes to which they give rise assume a certain appearance of kinship by virtue of this localization. It is always relatively easy, even for a beginner, to determine whether a cutaneous affection is follicular or not; it is far less simple, when dealing with a folliculosis, to distinguish its pathological character. This consideration in my opinion fully justifies the arrangement adopted in the following:

Pilo-sebaceous Follicles.—The pilo-sebaceous follicle is an invagination of the epidermis; its floor, raised as a papilla, gives insertion to the *hair*, which is secreted by this papilla; a lateral diverticulum, the *sebaceous gland*, secretes a fatty material, the sebum.

This follicle, of epithelial structure, projects into the derma, its extremity sometimes reaching the hypoderm. It is encased in a fibrous follicular sac, into which a muscle with smooth fibers, the erector pili, is usually inserted.

Two details in the structure of the follicle are of special interest for the dermatologist.

The first concerns the difference in its constitution above and below the opening of the sebaceous gland. Whereas in the deep portion the invaginated epidermis undergoes various modifications which transform it into the epithelial sheaths of the hair, it preserves in the external, less extensive portion, exactly the same characteristics as on the surface of the skin. This superficial portion, the follicular collar, also known as the *ostium* of the follicle, the *pore*, or on account of its usual form, the follicular *funnel*, participates therefore in the pathology of the surface epithelium.

Moreover, the follicular pore is especially exposed to repeated traumatism, through rubbing and scratching, because the hair which traverses it acts as the arm of a lever.

Finally, it represents an ever-ready receptacle for dust, irritative

matter of occupational, pathological or therapeutic origin and notably for microörganisms, pathogenic or saprophytic, to which it offers shelter. It has very properly been said that the follicular funnel is the flaw in the epidermic armor.

Another noteworthy anatomical fact is that the pilo-sebaceous follicle is surrounded by a network particularly rich in bloodvessels and nerves; with the result that the phenomena of reaction are easily elicited and well-marked.

The pilo-sebaceous follicles are distributed, in variable numbers and dimensions in different regions, over the entire integument, with the sole exception of the palmar, plantar and ungual regions and the semimucosæ. It is therefore an incorrect expression to speak of areas provided with fine downy hairs as glabrous or smooth, in contradistinction to hairy regions supplied with coarse hairs.

Folliculoses.—The affections of the follicles, which I group under this heading, are very numerous and variegated. Some concern the localization of a skin disease which occurs elsewhere, while others are pathological conditions peculiar to the follicles.

In the former case, the follicular localization may be accidental, as it were, without lending a special feature to the dermatosis; this is the case, for instance, in follicular purpura, in psoriasis, in soft chancre, etc. It seems to me superfluous to dwell upon these possible peculiarities.

In other cases, the follicular localization of the dermatosis is *elective* and visibly modifies the clinical picture; the affections of this kind (for example: follicular pyodermatitides, follicular syphilides, etc.), must necessarily figure in this chapter.

However, the preceding data do not furnish a sufficient basis of classification. The mode of grouping the folliculosis which seems to me most in conformity with clinical facts, distinguishes between:

1. *Acute suppurative folliculitides.*
2. Follicular complications of kerosis, namely *seborrhea* and the various *acnes* which result from the same.
3. *Depilating folliculoses.*
4. *Subacute folliculoses,* among which belong the follicular localizations of eczematides, syphilides and tuberculides.
5. *Pityriasis rubra pilaris,* which one is inclined at present to connect with the preceding group.
6. *Follicular keratoses.*

ACUTE SUPPURATIVE FOLLICULITIDES.

These are subdivided into two groups: those which are purely *pyococcic,* namely due to the ordinary microbes of suppuration; and those which are *trichophytic.*

They manifest themselves in the form of pustules pierced by a hair through their center, more or less superficial or deep, of variable size and surrounded by a congestive halo.

It has been shown above that Bockhardt's impetigo or staphylococcic impetigo, tends to assume the form of superficial and intra-epidermic osteofollicular pustules; it is unnecessary to repeat its description.

Not uncommonly the superficial suppuration is accompanied or complicated by the formation of a minute abscess, situated more deeply along the follicle and due to the penetration of the pyogenic microbe (Plate II): this condition is notably present in the case of folliculitides of the regions with coarse hairs, known under the old name of *sycosis* (from $\sigma\bar{\upsilon}\kappa o\nu$=a fig.). There are two varieties of sycosis: one simple or pyococcic, the other trichophytic; the latter will be described together with the trichophytic folliculitides.

Furuncle and carbuncle are acute staphylococcic folliculitides, deep from the start, with extensive inflammation, terminating in partial necrosis and suppuration. The primary follicular localization, sometimes very apparent, is less evident in other cases. The reader is referred for their description to the chapter on the pyodermatitides.

Acnes are often pustules, but they represent the inflammation of a preliminarily altered follicle and will be discussed in a subsequent section.

Sycosis Simplex.—The acute suppurative staphylococcic folliculitis of hairy regions, more particularly the beard, give rise to a symptom-complex, commonly designated under the name of sycosis simplex, vulgaris, or non-parasitic, in contradistinction to trichophytic sycosis.

Aside from the moustache and the beard, simple sycosis is met with at the pubis and in the axillæ in both sexes and on the hairy scalp in children.

The peripilar pustules, deep from the start or becoming so later, are scattered irregularly or they may be agminated. Sometimes they begin as follicular papules or as tuberous elevations or as more or less deep and extensive soft infiltrations, causing a sensation of tension, heat or shooting pains and suppurating only secondarily; often the suppuration is primary and early. The affected regions become covered with yellowish or brownish crusts, under which the skin is reddened, eroded and thickened; pressure upon them causes pus to escape from the enlarged follicular orifices.

A long while before falling out spontaneously the hairs can be pulled out easily and painlessly with forceps or with the fingers; they come out with their root surrounded by a gelatinous, translucid or opalescent sheath, which is their epithelial sheath infil-

trated with pus. This sign, which is typical of an involvement of the deep portion of the follicle, is absent in osteofollicular impetigo; it suggests that the papilla may be destroyed by the suppuration, with the result of permanent alopecia, although this is usually not the case.

Simple sycosis, following upon impetigo, furuncle, panaris, coryza or an infection by means of the razor, is spread from one place to another and carried to a distance by means of the fingers or toilet articles [especially towels]. The deep, intradermic seat of the affection, moreover, renders it rather inaccessible to therapeutic agents, which explains its obstinate, frequently chronic character and its tendency to recurrence. Cases of sycosis of the beard of many months' or even years' standing are met with.

The topography of the lesions permits a separation into several clinical types:

Sycosis of the beard (*mentagra of Alibert*), usually symmetrical, occupies the lower portion of the cheeks, whence it spreads upward toward the temples and downward to the subhyoid region, sometimes reaching the chin.

Sycosis of the mustache, often situated laterally under one nostril before becoming bilateral, is practically always connected with a lesion or infection of the corresponding nasal fossa. This manifests itself by chronic coryza, a habitual mucous or mucopurulent nasal discharge and ordinarily gives rise at the same time to folliculitis of the nasal fossæ and ciliary blepharitis or phlyctenular conjunctivitis through an ascending infection. It is noteworthy that trichophytic sycosis, on the contrary, is extremely rare in the region below the nostrils.

The beard and moustache may be invaded, as a whole, in strumous or exhausted individuals, convalescents from serious diseases or even in apparently healthy men in whom all pus infections find an especially favorable soil and tend to last indefinitely in consequence of an unexplained predisposition.

In the pubic or axillary region the suppurative folliculitides develop especially under cover of uncleanliness and neglect and sometimes after scabies. Often, as also in the case of the beard, they become superimposed on a seborrheic or artificial eczema.

The condition sometimes described as *eczema pilaris* is merely an eczema of the hairy regions complicated by a peripilar impetiginization or by staphylococcic folliculitis.

On the *hairy scalp* the folliculitides are observed chiefly in schoolchildren. Pediculosis and various traumatisms are common causative factors. There may be a rapid and profuse outbreak of suppurative folliculitis over a part of the scalp or over the entire scalp, as the sequel of removal of hairs with the forceps, painting with

iodin tincture, application of salves, etc., in the course of treatment, for instance, of an attack of tinea.

The *treatment* of sycosis vulgaris is likely to be tedious. In the first place the diagnosis must have been positively confirmed by the microscopic examination of the hairs, showing the absence of the trichophyton.

The rule is to stop the use of the razor, to cut the hairs short with scissors, to clean the crusted surfaces with sprays or moist dressings, to pull out the hairs which are easily detached, to empty the pustules and pack them several times daily with absorbent cotton soaked in "eau d'Alibour" or in resorcinated or camphorated alcohol.

At the onset, starch poultices are better tolerated than salves or pastes containing calomel, yellow oxide, ichthyol, sulphur, salicylic acid or resorcin, etc., which may be indicated later on.

Total epilation is sometimes indispensable, notably when the suppuration has reached the depth of the follicle. Preliminary radiotherapy greatly facilitates this and by itself alone often acts very favorably, even in weak doses.

In obstinate cases, vaccinotherapy is often advantageously employed, either with stock staphylococci or with an autogenous vaccine. Good results are sometimes obtained through the application of zinc pastes with oil of cade and sulphur or with ichthyol (5 per cent.) containing resorcin (3 to 5 per cent.).

In the terminal stage, mercurial plasters, sometimes scarifications and the *x*-rays are effective in the obliteration of the indurations. Correction of hygienic conditions and general tonic treatment must not be neglected; beer-yeast, ferments, arsenic and sulphur water may be of use.

It is needless to say that from the start everything must be done to extinguish the original foci of the pyococcic infection and especially to cure the chronic coryza in cases of sycosis of the moustache.

Trichophytic Folliculitides.—The trichophytons which cause suppurative follicular inflammations are ectothrix trichophytons, that is, they vegetate in the sheath of the hairs and not or very slightly in their interior. Adults are rather more susceptible than children. The parasites are directly or indirectly of animal origin.

Several clinical types of these trichophytic folliculitides may be described:

Sycosis trichophytica barbæ is due, in the majority of cases, to the trichophyton of horses (trichophyton gypseum); it grows in white cultures and is in itself highly pyogenic. It gives rise to peripilar pustules, with a fairly intense inflammatory zone and a rapidly swelling base; they become agglomerated in red and raised, tuberous, firm or even indurated patches or masses from which pus

escapes through numerous orifices on pressure; sometimes purulent sloughs occur. These patches gradually extend peripherally and new regions at a distance are attacked. The hairs in the middle of the patch easily come out with the forceps and are denuded and dead; it is necessary to look at the margins for hairs still provided with their root-sheath, where it is easier to find the parasitic spore-bearing mycelium under the microscope.

The lesions are preferably situated in the lower portion of the cheeks or the chin, sometimes at the temples or in the hyoid regions (Fig. 121); they are often asymmetrical; in one case I found them on the moustache, an absolutely exceptional location.

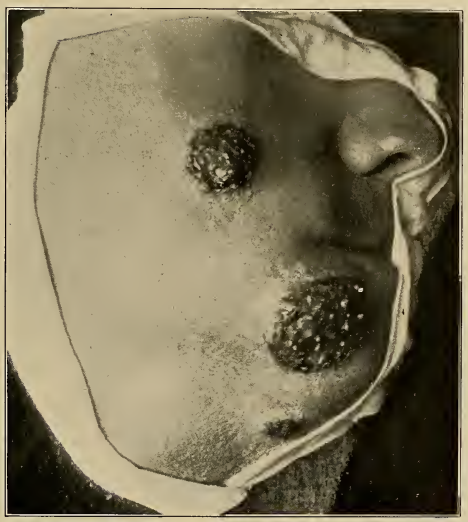


Fig. 121.—Trichophytic folliculitis of the beard; parasitic sycosis due to trichophyton ectothrix. After a cast in the Museum of the St. Louis Hospital, Paris.

Trichophytic sycosis is observed especially in coachmen, grooms, knackers, veterinaries and horseshoers.

Trichophytosis of the beard may assume other aspects; that of drier, less suppurative papules or tuberosities; that of red circles or arcs, with white scales and dry epidermic plugs, from which a broken hair emerges, suggestive of keratosis pilaris, etc. This is undoubtedly due to a difference of the trichophytic species; as a matter of fact, a bird trichophyton with rose-colored cultures has

been demonstrated, a yellow species, the acuminatum, the violaceum and so forth.

Kerion Celsi is the name applied to patches of trichophytosis occupying the hairy scalp of children or adults and similar to those described above in the beard; they are likewise generally due to the trichophyton gypseum.

The condition, as a rule, consists of one or several nummular or larger, distinctly elevated, rounded disks, with sharply outlined borders of a bright red color; the hairs have fallen or are easily detached; there is a scattering of small white pustules which can be emptied by pressure.

Folliculitis Agminata, or Leloir's *folliculite conglomérée en placards*, is the same affection as kerion, but occupies the smooth parts. It is observed at all ages, on the wrists, the forearms and the neck. The red patch, comparable with a macaroon, is covered with a crust or with thick pus; after it has been cleansed it appears interspersed with dilated follicular orifices which riddle it like a sieve. There may be several patches of different ages. Their growth is rapid and takes place in a few days.

The spores and fragments of the mycelium are often difficult to demonstrate in the pus, so that the nature of this affection was not recognized by Leloir. Like kerion, it sometimes lasts for weeks and months, but it may disappear spontaneously.

The suppurative trichophytic folliculitides leave, as a rule, more or less alopecic cicatrices.

The *diagnosis* can sometimes be based on the clinical characters alone, but should always be confirmed by microscopic examination and if possible by culture. The pyococcic folliculitides, even when agminated, do not form as distinctly circumscribed round patches and are more apt to be disseminated. Furuncle and carbuncle have a more pronounced and extensive inflammatory edema, are more deeply infiltrated and much more painful.

The classic *treatment* of the trichophytic folliculitides, no matter where the lesions are situated, consists of the careful epilation of the patch and of a peripheral zone of about 1 cm. in width; as the hairs are not brittle, radiotherapy may be replaced by avulsion with the forceps, followed by the application of emollient or antiseptic dressings. It is sometimes necessary to open the deeper follicular abscesses with the thermocautery. Finally, the affected surfaces are freely painted with tincture of iodin every other day, or they may be dressed with iodin in vaseline.

However, all dermatologists have noted that very freely suppurating trichophytoses heal spontaneously, as it were; thorough epilation, cleansing and moist dressings or starch poultices are sufficient. This fact is explained either by an immunization or more probably

by the expulsion of the parasites through the suppurative reaction induced by them.

The precautionary measures necessitated by the contagiosity of this affection must, of course, be insisted upon.

SEBORRHEA.

The word seborrhea, which means a flow of sebum, was introduced in 1840 by Fuchs to designate the condition named *acne sebacea*, by Biett; *sebaceous flux*, by Rayer and *steatorrhea* by E. Wilson.

I have stated above that, in my opinion, seborrhea is one of the principal manifestations of a more general diseased condition which I have named kerosis.

The meaning of the term seborrhea has been unreasonably extended by authors to the point of applying it to all the other manifestations of kerosis, and even, by Hebra, to pityriasis simplex, thereby leading to the enormous confusion which still prevails as illustrated by the commonly used but highly improper terms of *seborrhea sicca* (for pityriasis simplex), *seborrheic eczema* (for eczematide) and so forth.

Seborrhea is an exaggerated sebaceous secretion. A distinction can be made between fatty seborrhea and oily or fluid seborrhea; but there are numerous intermediate conditions.

Fatty seborrhea [seborrhœa steatosa] is characterized by the dilatation of the pore and the "collar" of the follicles, especially those with which the larger sebaceous glands are connected, with accumulation in the osteo-follicular canal of a substance composed of horny cells, fat and microbes, namely the *sebum*.

Sometimes the horny cells predominate and are concentrically arranged, forming the *seborrheic utriculus* or *seborrheic cocoon* of Sabouraud; a somewhat greater amount of hyperkeratosis results in the comedo of acne.

In other cases, the fat forms with the horny cells a pasty whitish substance, of a butyric acid odor, which can be expressed by squeezing between two fingernails, under the aspect of a worm or vermicelli; this is the *seborrheic filament*.

In order to make sure that the condition is really one of seborrhea, instead of a dry follicular keratosis, for example, it is therefore necessary to demonstrate that the fatty material under consideration can actually be pressed out from the dilated follicular orifices. This may be done either by compressing the skin between the nails, as mentioned above, or by scraping it forcibly with a glass slide or a blunt scalpel. The substance thus obtained must be fatty; the cocoons or filaments are easily crushed. Under the microscope are seen fat drops and horny cells or débris of horny cells;

moreover, on staining the specimen with an aniline dye, preferably with thionin, an enormous number of microbes become visible.

Credit is due to Sabouraud for having shown that these microbes, myriads of which exist in the product of fatty seborrhea, belong to a single species the *microbacillus of seborrhea*. This is very small, barrel- or rod-shaped, often curved, and not easily grown in cultures. Hallé and Civatte have demonstrated in my laboratory that it grows much more readily and abundantly in anaërobic cultures. This bacillus was seen by Unna and Hodara, who interpreted it as the microbe of acne.

According to Sabouraud, its extreme abundance in pure cultures indicates its pathogenic nature, so that seborrhea should be interpreted as a parasitic disease. This theory is not acceptable, and it is more likely that this very widespread microbacillus becomes implanted whenever it can live and that its growth is secondary to the seborrhea.

Seborrhea oleosa [or *fluxus sebaceus*] manifests itself, in its milder degrees, by a greasy and shining state of the skin, which leaves fat spots on paper; in the advanced degrees, actual drops of oil are seen to dot the integument. It almost invariably coexists with fatty seborrhea. It is very difficult to decide if the fluid fat is really derived from the sebaceous glands instead of from the sweat glands; namely, if the condition is not one of *hyperidrosis oleosa*.

The usual *distribution* of fatty seborrhea is less extensive than that attributed to it by those writers who confuse it with kerosis. Its seat of election is in the center of the face, on the alæ of the nose and in the nasogenial grooves; it is less common in the other regions of the face and on the vertex, rather rare on the thorax and the genitals, exceptional in other kerotic regions. Oily seborrhea is observed on the face, on the hairy scalp and sometimes on the thorax.

Its *etiology* merges with the etiology of kerosis. It is practically never observed before puberty. Its relations with sexual development, genital disturbances and gastric affections are very evident.

The *treatment* is also that of kerosis. Locally, less reliance should be placed on fat-removing washes and lotions, containing ether or other ingredients, than on sulphur or camphor, etc. Systematic massage of the skin favors the evacuation of the retained products; its exaggerated employment would be injurious.

THE ACNES.

The term acne has been applied, since Willan, to all eruptions which were believed to be due to an affection of the sebaceous

glands; the clinical appearance, or the related cause or probable character of this affection being specified by an adjective. A reaction has set in against this linguistic abuse which led to confusion.

Seborrhea is no longer described as *acne sebacea* or *oleosa;* we speak of keratosis senilis instead of *acne sebacea concreta; acne miliaris* or *milium* is a form of epidermic cyst; the *acne varioliformis* of Bazin, or molluscum contagiosum, is an epithelial tumor; *acne rosacea* belongs to the chronic erythemas and its complication; *acne hypertrophica* to the hypertrophic dermatoses; *acne cornea* is a keratotic folliculitis; *acne decalvans* is a decalvating folliculitis; *acne cachecticorum* is a variety of tuberculide as *acne syphilitica* is a form of syphilide.

There remains a dermatosis which is typical of the genuine acnes, namely acne vulgaris or juvenilis; and, in addition, a few related varieties.

Acne Vulgaris or Juvenilis.—This is a very frequent complication of kerosis and of seborrhea in particular, manifesting itself as a regional, successive, follicular eruption, especially affecting youthful individuals.

Acne is not characterized by a single eruptive lesion, but by a polymorphous set of lesions more or less derived from each other; comedones, papulo-pustules, superficial or deep follicular pustules, indurated abscesses, crusts and cicatrices. Besides the cases with perfectly developed lesions, there are very numerous individuals suffering from incomplete acne, with a few comedones and, from time to time, a papulo-pustule.

A *comedo* is a small horny mass, with a brown or black top, the size of a pinhead to that of a millet-seed, imbedded in a dilated follicular orifice where it resembles a powder grain. It can be squeezed out by pressure between two finger-nails, in the form of a firm yellowish mass with a black head, followed by a white unctuous filament, resembling vermicelli or a black-headed worm. Sometimes, "double comedones" are encountered, meaning comedones situated very close together, with a communicating base.

The comedo results from an ostiofollicular hyperkeratosis; its configuration is that of a small cylinder formed by concentric horny lamellæ; its exposed surface is colored, not by a deposit of dust, but through oxidation of the keratin itself; the cavity from which it may be expressed contains microbacilli in large numbers and sebum. It resists more or less the escape of the sebum, which is retained below; in the face, the *demodex folliculorum* is not infrequently met with.

Comedones in variable number, at first imperfectly formed and hardly distinguishable from seborrheic cocoons, later more voluminous, occur preferably in the face, especially on the nose, the

cheeks and the temples, on the chin, the back, the chest and the shoulders and rarely elsewhere.

When they exist alone they constitute *acne punctata*, the "blackheads" of the vernacular.

A little redness and tumefaction around some comedones are characteristic of *papular acne*. The inflammation is almost invariably more intense; the red acuminate elevation, the size of a pinhead to that of a pea, whitens at its apex in two or three days, due to suppuration, the pus may be evacuated or dry up as a crust while the papule collapses, becoming transformed into a brownish-red spot which leaves a minute cicatrix. This constitutes *superficial pustular acne*. It is more or less discrete or confluent and occupies the face, the shoulders and the thorax.

The pustules develop practically without pain and with hardly a little itching.

When the papulopustules have the size of a large pea or a bean and are hard, purplish and painful, suppuration being slow but deep and abundant, the condition is described as an *acne tuberosa* or *indurata*. In the form known as *phlegmonous acne*, the dusky red and fluctuating elevations surmount real acneiform abscesses, dermic or hypodermic; sometimes there are cavities with oily contents.

These different varieties are often associated in the same patient (*polymorphous acne*) in variable proportion (Fig. 122). In severe cases, the face, chest and back may be covered with lesions in all stages of development and with countless cicatrices of variable size, producing real deformities, so that almost no healthy skin is left. Sometimes the eruption has a tendency to become localized in one of these regions.

The topography of acne is very specific; it practically never passes below the belt line, nor the upper two-thirds of the arms, nor the margin of the scalp, where it invariably stops.

The eruption is maintained through uninterrupted crops of new lesions; it is continuous, but with periods of exacerbation in the spring, at the time of menstruation or in connection with errors in diet. [It constitutes about $8\frac{1}{2}$ per cent. of all cases seen in dermatological practice in Ameria.]

Etiology and Character.—The condition *sine qua non* of acne is kerosis; individuals having acne always suffer from seborrhea, pityriasis simplex and sometimes from eczematides. A predisposed soil is present and persists beyond the acne.

The eruption begins in both sexes at the approach of puberty, flourishes toward the sixteenth and eighteenth year and diminishes between the twenty-second and the thirtieth year; it is not uncommonly followed by rosacea, baldness or eczematides.

The causes of kerosis accordingly predispose likewise to acne; in the first place, the molimina, genital excitement, functional or organic genital disturbances. A very correct observation, frequently made, is that acne localized on the chin of young girls or young women almost certainly indicates some utero-ovarian ailment.

Digestive disturbances, improper diet, gastric dyspepsia and habitual constipation play an equally important part.

A pale complexion, a "lymphatic" or "arthritic" constitution, are often met with in acne patients, especially those with a kerotic heredity.

Fig. 122.—Juvenile polymorphous acne.

As to the determining cause of the eruption, that which transforms a seborrhea into acne, it is practically certain that this is primarily of local microbic, infectious origin. This is not an established fact for comedo, although Sabouraud has shown that the comedo contains in its interior myriads of seborrheic bacilli (the old acne bacillus of Unna and Hodara), and that in its surroundings the bottle-bacillus and cocci are usually present.

The acne pustule almost regularly encloses, sometimes in enormous numbers, staphylococci of various kinds, which are pyogenic; the pustule is only rarely sterile.

From the benign character of this suppuration, its freedom from pain, its slow development, it may be inferred that the ordinary causative agent is not a virulent staphylococcus, such as the Staphylococcus aureus, but more probably the Staphylococcus albus or the staphylococcus growing in gray cultures, the polymorphous coccus of Cedercreutz, or an analogous organism.

Summarizing, the acne pustule is a folliculitis and perifolliculitis of a follicle obstructed by a comedo and infected by a pyococcus of moderate virulence. The abscess, resulting from the onrush of leukocytes, is primarily situated in the wall of the follicle, under the comedo, destroys this wall in a part at least of its circumference and more or less deeply invades the perifollicular tissue; this explains why it leaves a cicatrix. The sebaceous gland plays only a very subordinate part in the process.

Treatment.—Both local and general treatment are required.

Locally, the skin must be kept very clean by means of baths and warm soapy or alcoholic lotions. Camphorated alcohol, at first dilute, camphor and sulphur lotions, sulphuretted or mercurial washes, or chlorhydrate of ammonia, yield much better results than salves of any kind. Mild measures must be employed to begin with, for the skin in some cases of acne is extremely irritable.

After washing with soap, before applying the selected lotion, it is advisable to squeeze out the comedones from time to time, by systematic massage, pressure with a watch-key, or even by means of a special "comedo punch" and to open the superficial pustules with a flamed needle. These procedures must not be overdone, however, as the eruption is occasionally aggravated in this way.

In severe and obstinate cases, the rapid exfoliating method is recommended; I have repeatedly been enabled to obtain nearly complete and durable cures with it. Gradual exfoliation is often equally successful, for example with the "strong salve" of Brocq (see Therapeutic Notes) which is allowed to act from five to thirty minutes before a lukewarm wash or bath. The irritation is then soothed by means of a zinc paste.

In case of indurated or phlegmonous acne, the galvanocautery, if necessary the thermocautery, serve to evacuate the purulent collections and suppress the developing lesions. Radiotherapy, employed as a last resort, sometimes yields very valuable results.

The *internal treatment* must aim in the first place at the correction of the general hygiene. The diet must be closely regulated, omitting all stimulants, sweets of all kinds, fermentable substances and an excess of meat. The physician must see to the regularity of meal hours, good mastication, the condition of the teeth, regular evacuation of the bowels, which will sometimes have to be secured

by suitable laxatives. Disturbances of the genito-urinary apparatus must be corrected as far as possible. Exercise, friction of the cutaneous surface and massage contribute to regulate the general circulation.

L. Jacquet advocated, besides bradyphagia, "massage of the face by pétrissage" and facial gymnastics; these measures often prove really efficient.

The remedies to be recommended are, according to the cases and sometimes successively, arsenic, cod-liver oil, ferruginous agents, ichthyol, beer-yeast and lactic ferments; the latter are sometimes highly successful, sometimes entirely inefficient. Staphylococcic vaccintherapy, or better, inoculation with a mixed autovaccine, may likewise have excellent results or in other cases prove useless.

[In my experience, juvenile acne may be successfully treated by local measures alone without any regard to the general health. Proper exercise, regular bowel movements and a diet free from excesses of all kinds are good for every one but I have never been able to clearly establish a relationship between juvenile acne and any general abnormalities whatever. One of the most severe and most obstinate cases of acne I have ever encountered occurred in an otherwise perfect specimen of young manhood who lived at the trainer's table and observed the severe regimen required of a member of a college athletic team.

My plan of treatment consists of various measures of local antiseptics: washing with soap, *avoidance of friction* with the towel which serves to spread infection, soaking ten to fifteen minutes with a 1 to 2000 weak alcoholic watery solution of bichloride of mercury (the soap must be thoroughly rinsed off first) and, after washing away the bichloride solution and drying, an application of sulphur paste, *i. e.*, zinc oxide ointment containing 10 per cent. of kaolin and 10 per cent. of precipitated sulphur, to be applied at bed-time. In the morning a soap and water wash. The keratolytic action of the paste becomes obvious after five to ten days; the skin feels tight, drawn, as if coated with varnish. This symptom is the signal for omitting the sulphur paste one night and applying instead a soothing and softening cream consisting of 2 per cent. salicylic acid in cold-cream or petrolatum. The next night the paste may be resumed and thereafter must be interrupted by one night of the cream every fourth or fifth day. At the same time it is of primary importance to remove mechanically as many sources of infection as possible; comedones must be squeezed out and pustules evacuated systematically at intervals of three, later seven to ten days. My own experience with vaccines has not been favorable; others, notably Gilchrist and Engman have reported excellent results.]

Medicinal and Occupational Acnes. –These are closely related to juvenile acne and if proof were required, would show the part played by the ingesta as well as by external irritants in the genesis of this skin disease.

The iodides and, to a less degree, the bromides produce in some individuals, either from the start or after prolonged administration, an iodide or bromide acne; its special features are the age of the patients, the sudden onset and the inflammatory indurated nodular character of the eruptive lesions. These acnes are localized chiefly on the face and the back and may become associated with other symptoms of iodism or bromism.

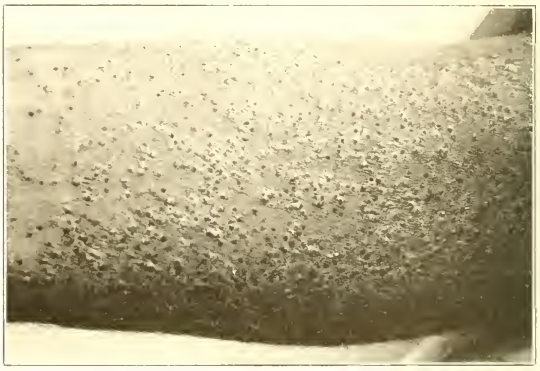


Fig. 123.—Cade oil acne; internal aspect of the right thigh in a case of psoriasis treated with an oil of cade glycerolate. The pustules contained the Staphylococcus aureus; cured by means of a soapy salve.

The various preparations of tar, oil of cade in particular, give rise in those who handle them or apply them to the skin (in the treatment of psoriasis, for example) to an eruption of reddish-brown papulopustules, centered by a comedo, very much like acne (Fig. 123).

Mineral oils, employed for the lubrication of engines, may cause either acute suppurative folliculitides or subacute acneiform folliculitides, analogous to tar acne. All these hydrocarbons act apparently by producing an ostio-follicular hyperkeratosis, which retains the pyococci and creates conditions favorable to their growth and to an inflammatory reaction of the integument.

Chlorin acne presents the features of juvenile acne, although greatly exaggerated, with an extraordinary profusion of comedones and a general distribution over regions which escape in common

acne; it has been observed in laborers employed in sodium chloride electrolytic works. According to Fumouze (1901) it is due to nascent sodium hypochlorite.

Acne Necrotica of Boeck (*Acne Pilaris of Bazin, Acne Varioliformis of Hebra, Frontalis, Rodens, etc.*).—This is a disease affecting middle-aged and old persons and presents characteristic lesions.

The eruption consists of pink or waxy papules the size of a pinhead to that of a small lentil, whose apex very rapidly assumes a yellowish hue, simulating a vesico-pustule and dries to a biconvex brownish-yellow crust; this very adherent crust is embedded in the skin and encircled by a sometimes slightly raised border; its fall is often delayed and leaves an indelible depressed cicatrix.

Advancing in continuous or intermittent crops, the eruption is situated on the forehead, at the border of the scalp which is often in part invaded, at the temples, in the conchæ of the ears, sometimes on the nose and in the nasogenial grooves and very rarely on the middle of the back and the chest. The lesions are grouped or disseminated. The affected regions finally become riddled with cicatrices. When left untreated, the affection lasts for years and even indefinitely.

The histological lesion consists of a dry lenticular necrosis, situated at the orifice of a follicle and comprising the entire thickness of the epidermis and a superficial portion of the cutis; surrounded by an inflammatory zone with thrombosis of the bloodvessels.

Necrotic acne is distinguished from crusted secondary syphilides by its necrotic character and its cicatrices; from tuberculo-crusted tertiary syphilides, by the absence of induration and by the dissemination of the lesions.

Only the microbacillus of seborrhea and various staphylococci, especially the aureus, have ever been discovered in this acne; it is probable that another etiological factor is involved, but it is unknown. Kerosis and seborrhea constitute the necessary soil.

Appropriate treatment ensures a cure in a few days. The employment of soap and water and of a strong sulphur-cade- and salicylic-acid salve is sufficient; all internal medication is unnecessary. Sulphur washes or a sulphur salve are recommended for the prevention of recurrences. [I have found soap and water and the application of ammoniated mercury ointment, which is not unpleasant, quite sufficient for a cure.]

Keloid Acne.—This affection, first described by Bazin—identical with the *dermatitis papillaris capillitii* of Kaposi—occurs essentially in males and is almost exclusively observed at the *nape of the neck*, at the border of the hairy scalp and very rarely in the beard.

It begins as an acuminated papular folliculitis, the size of a millet or hemp seed, which may become pustular; originally disseminated,

the lesions multiply and become grouped, finally coalescing into a band which occupies the entire posterior border of the scalp.

This folliculitis is distinguished from the start by its subacute inflammatory character, its very marked induration and a course leading to the formation of fibrous excrescences.

The confluence of these lesions results in a horizontal row of agglomerated fibrous tubercles and finally a "cushion" which may have the thickness of a finger, a length of 10 to 15 cm., or even extend from one ear to the other. Cicatricial and smooth on its lower aspect, this cushion bristles on its upper side with hairs arranged in tufts or wisps. On pulling these out, one is surprised by the depth of their implantation.

The duration of the affection is indefinite; it may be prolonged for fifteen to twenty years, or longer. The cushion slowly extends upward on the occipital region, never in a downward direction, and leaves behind it a pinkish, more or less thickened permanent cicatrix.

Histology shows a chronic follicular inflammation, noteworthy in that the cellular infiltration is almost exclusively composed of plasmocytes, with a few giant cells; it leads to the formation of a dense hyperplastic fibrous tissue; the process is absolutely different from that of the true keloids, as shown especially by Pautrier and J. Gouin. Its nature is not known and the special microbic agent which is probably present has not been demonstrated. The coexistence of kerosis is invariable.

Keloid acne is very obstinate to *treatment*. Antiseptic washes, iodin applications, mercurial ointments, sulphur, naphthol and tar salves are indicated only in the first stage and are rarely sufficient. The best obtainable results are secured by the following method: First, all the active lesions of folliculitis are destroyed with a fine-pointed thermocautery; then the keloid growths are attacked by very deep and repeated scarifications, followed eight or ten days later by a number of radiotherapeutic sessions.

In well-marked cases, surgical removal is advantageously performed, followed by radiotherapy. Extirpation alone is not to be recommended as it is often followed by recurrence.

CICATRICIAL DEPILATING FOLLICULITIDES.

The nosographical group of depilating folliculitides, or acne decalvans, is indefinitely outlined and its varieties are still imperfectly formulated for lack of knowledge of the etiological factors. All the deep folliculitides may become depilatory through permanent atrophy of the follicle, as in furuncle, favus, keratosis pilaris, sycosis, the acnes and the syphilides. But those under present consideration are inflammatory, sluggish, progressive, rebellious,

often agminated and necessarily leave true cicatricial tissue behind them; they are the forms which lead to the cicatricial alopecias.

Pseudo-pelade of Brocq (*alopecia atrophicans* of foreign authors) represents the most typical form. It is characterized by alopecic and cicatricial spots or patches, scattered or grouped on an otherwise healthy scalp. These spots have various dimensions and shapes, being sometimes barely lenticular, rounded and very numerous; sometimes several centimeters in diameter, irregular, with geographical outlines; or the two varieties may be associated (Fig. 124). The borders are distinct, without a transitional zone; the surface is wax-white or faintly pink, perfectly smooth, without scales or broken or lanugo hairs; even the follicles have disappeared, as may be ascertained by painting the surface with tincture of iodin.

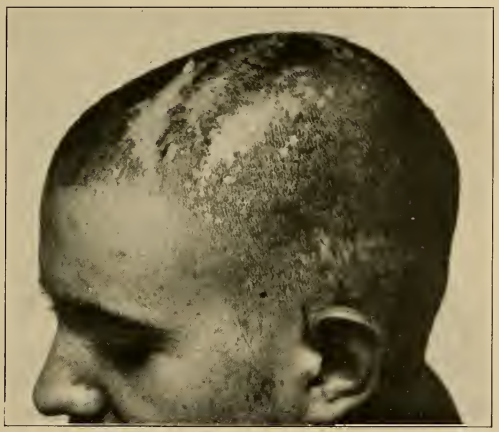


Fig. 124.—Pseudo-pelade of Brocq.

This affection is met with on the hairy scalp of youthful or adult individuals, especially in men and rarely in the beard. The medium-sized and large spots are evidently the result of growth and confluence of small spots. The onset is insidious and usually escapes notice; Dreuw believes that it takes place in childhood as an *alopecia parvimaculata*.

In watching the progress of the affection, which is extremely slow, it is seen to begin with a slight orificial keratosis at one or several hairs surrounded by a pinkish area, at the border of the patch; next follows the definite loss of these hairs and the extension

of the cicatrix to this area. Despite all efforts and despite the probability of a parasitic cause no special fungus or microbe has ever been found in the hairs or in the follicles. Under the microscope a cellular infiltration is seen around the follicles and around the blood-vessels and lymph vessels which are greatly dilated; the process terminates in a connective-tissue and elastic atrophy. It is certain that pseudo-pelade must be considered as a folliculitis, but it will be noted that the follicular inflammation is clinically not very evident, far less so than in the following forms.

The differences between pseudo-pelade and ringworm are considerable. There is a much greater analogy with the cicatrices of favus, so much so that in my opinion the disease would be better designated *pseudo-favic alopecia*, but there have never been yellow pits or scutula, the cicatricial skin is not so red, the hairs are not dull and lusterless and there is no fungus. The patches of lupus erythematodes are less numerous and the process extends in the form of very red and hyperkeratotic spots.

By means of sulphur lotions, sulphur, cade, naphthol, resorcin salves, mercurial ointments, etc., one may hope to arrest the progress of the disease, but not to restore the hairs whose follicles have been destroyed. The treatment should therefore begin as early as possible.

Folliculitis Decalvans.—*Folliculite épilante* of Quinquaud—*acne decalvans* of Lallier—is perhaps merely a variety of the preceding type from which it differs only by the presence in the invaded zone of a few scattered pustular follicles, the size of a pinhead or a small pea.

Lupoid Sycosis of Brocq.—*Lupoid acne* of American writers, *ulerythema sycosiforme* of Unna, *dermatitis sycosiformis atrophicans* of Ducrey and Stanziale, is situated on the beard and especially on the cheeks (Fig. 125). It gives rise to agminated follicular pustules, with inflammatory redness and diffuse superficial infiltration of the skin.

It differs from ordinary sycosis by its tendency to extend uniformly and by the red, smooth, central *cicatricial* alopecia, often of a keloidal appearance, which it leaves behind. It may greatly resemble a flat lupus vulgaris in course of central cicatrization; but there are no lupomas and only suppurative folliculitis. The single focus, or at most two or three foci, in the course of years may attain the dimensions of the palm of the hand.

Depilating folliculitides of the smooth parts, described by Arnozan and Dubreuilh, which occupy the thighs and legs, are still more rare; they constitute a type related to the above or perhaps a form of tuberculides.

Furthermore, an entire series of cases of *necrotic* (Janovky) or

ulcerative (Bizzozero) *folliculitides* or *perifolliculitides* has been reported; their significance and nosological position have not been determined.

Treatment.—Washes with alcohol or sublimate, yellow oxide salves and mercurial plasters, combined with epilation, have long been the best means for combating the progress of lupoid sycosis and analogous affections. Radiotherapy is greatly superior to these measures and yields noteworthy results.

FIG. 125.—Lupoid sycosis.

SUBACUTE FOLLICULITIDES.

There exists a group of red folliculitides, of acute or rather subacute course, which are neither regularly suppurative nor necessarily depilatory.

Follicular Eczematides.—I employ this term for the *peripilar seborrhëids* of authors. The *acute form* manifests itself especially in markedly kerotic men, preferably at the beginning of the warm weather, in the form of an eruption of small red acuminate follicular papules, agminated in one or several patches on the back, the shoulders or the anterior side of the trunk. The patch extends by invasion of all the neighboring follicles, while at the center the eruption subsides and leaves behind a yellow slightly desquamating surface. The course is rapid at the onset. The diseased surface attains the size of the hand in the course of a week or two; later on it is sluggish.

A *subacute form* of this affection has been frequently described. It is characterized by small spots of a yellowish red, or a purplish red, perifollicular, barely papular, arranged in groups of ten to thirty, which gradually become more prominent and covered with a yellowish scale or crust (Fig. 126). Confluence of these lesions may lead to the production of spots of figured eczematides.

Their seat of election is, moreover, the same as that of the figured eczematides which may have preceded the follicular eruption. The latter is frequently seen also on the limbs, the thighs, the legs, the wrists, the forearms, etc. Pruritus is very slight.

Fig. 126.—Subacute follicular eczematides.

Treatment with sulphur baths, sulphur or ichthyol salves, is very promptly effective in the acute form; it must be more energetic or more prolonged in the sluggish varieties.

Eczema Folliculorum of Malcolm Morris and Unna is, I believe, merely a rare variety of the preceding affection. It consists of a non-suppurative folliculitis, agminated in small red patches, which are distributed on the trunk and especially on the limbs, progressing extensively, with a tendency to complete eczematization. Pruritus is sometimes rather troublesome and causes scratching. This form is somewhat rebellious to treatment.

Follicular Syphilides.—Among the papular secondary syphilides, there is a form with small papules, known as *miliary, granular, lichenoid*, or *acneiform peripilar syphilides*. They result from a localization of the syphilitic infiltration around and beneath the pilo-

sebaceous follicles; it usually seems to be invited by a preliminary lesion of these follicles, in the form of kerosis or of keratosis pilaris.

The follicular syphilides usually make their appearance from four to eighteen months after the chancre; this is therefore a secondary manifestation but not one of the earliest. A distinction is made between a papulo-squamous, papulo-pustular and even a vesicular

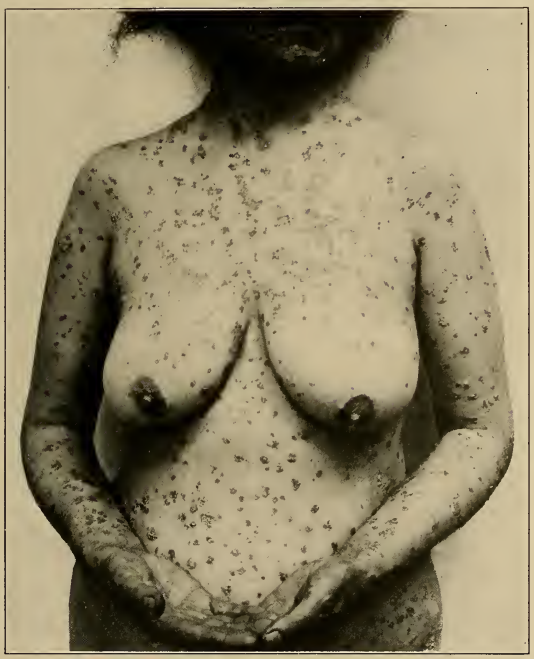


FIG. 127.—Follicular syphilides, papulo-squamous variety. On the face the eruption was distinctly papulo-lenticular.

variety. The eruption is disseminated but very often consists of small groups of agminated lesions (Fig. 127). Its seat of election is on the trunk, where it is seen on the back, flanks and loins; the limbs may also be involved.

The *papulo-squamous variety* consists of small miliary elevations, of a dusky red color, acuminate and crowned with a dry scale

which is enclosed in the follicular orifice and not easily removed. The principal features of these lesions are their firmness, so that they feel like granules to the finger and their relatively slow development; they persist several weeks without change.

The *papulo-pustular variety* may become combined with the preceding in the same eruption, or it may exist alone; it is often very diffuse and abundant. The dark-red perifollicular protuberance is larger than in the squamous variety, even lenticular in size; it is surmounted by a vesico-pustule, always containing much less fluid than one would expect, rapidly drying into a crust. Under the vesicle or the crust is seen a dilated, sometimes eroded follicular orifice.

According to the size and appearance of the papulo-pustules or papulo-vesicles, the terms of *acneiform*, *varicelliform*, *herpetiform*, *varioliform syphilides* have been employed (A. Fournier). There occur transitional cases between this variety and the papulo-crusted or ulcerative syphilides.

Whether squamous or pustular, these syphilides may in some cases become arranged in *rings*, or grouped in collections of about fifteen to at most fifty lesions, sometimes around a large lenticular or crusted papule. These *agminated efflorescences*, absolutely characteristic of syphilis, have been picturesquely described as *syphilides en bouquets* and *corymbiform syphilides*.

Careful study of the features of the eruption and the concomitant symptoms will guard against confusion of follicular syphilides with keratosis pilaris, follicular eczematides, pityriasis rubra pilaris, or with acne, papulo-necrotic tuberculides, etc. The differential diagnosis from lichen scrofulosorum may be much more difficult even with the help of a biopsy, for their structure is rather commonly tuberculoid, but there is a positive Wassermann reaction.

The follicular syphilides are tenacious, rebellious to *treatment*, often leave persistent brownish macules, and have a tendency to recurrence. Treated with mercury, they last six weeks or longer; by means of arsenobenzol they can be removed in two or three weeks.

Follicular Tuberculides.—I have previously discussed the lichenoid tuberculides with small acuminate and peripilar lesions, which belong under the heading of *lichen scrofulosorum*.

The *papulo-necrotic tuberculides* seem to be so evidently related to the follicles that their first description by Barthélemy was given under the names of *folliclis* and *acnitis*. Histological examination serves to confirm the clinical impression in this respect. It would, therefore, be justifiable to group them among the folliculoses; at any rate, the question of their differential diagnosis from follicular affections is constantly raised. Their follicular localization is really accidental, however; it is sufficient proof to point out that these

tuberculides very commonly affect the palmar region, where follicles are absent. The reader is therefore referred to another chapter for this description.

PITYRIASIS RUBRA PILARIS.

Devergie, E. Besnier and Richaud have described under this name a skin disease distinct from psoriasis, from genuine pityriasis rubra [Hebra] and from lichen planus, in spite of a few real analogies. On the other hand, Hebra and Kaposi have studied under the name of *lichen rubra acuminatus* an eruption resembling it to such a degree that at the International Congress, Paris, 1889, the identity of the two pathological types was admitted; Kaposi himself emphasized this identity. It is desirable, however, to reserve the name of lichen acuminatus only for the acuminate forms of Wilson's lichen, which are, however, very rare.

The characteristic lesion of pityriasis rubra pilaris is a small *squamous follicular papule.* It is of a bright or dull red color, or pinkish, sometimes colorless at first, prominent, of acuminate shape with a conical apex which bears a follicular orifice covered by an adherent, dry, white scale, enclosing one or more often atrophied and shrivelled lanugo hairs. These follicular papules are dry; they are never seen to be vesicular or pustular; their size is that of a pin-head or a millet-seed; they are firm to the touch and as a result of their distribution the skin takes on a granular appearance and has the feeling of a grater when scrubbed with the hand.

Isolated at first, these papules multiply and become agminated later on; the intermediate skin becomes reddened and there follow thickened spots, patches, or surfaces, of all dimensions, of a yellowish-pink color, covered with pityriasic or psoriatiform, sometimes granular scales, dotted with horny points or quadrilated and lichenized. Their borders are, as a rule, irregular, dentated and surrounded by characteristic peripilar papules (Fig. 128).

The *eruption* is usually distinctly symmetrical. Its distribution is fairly constant in fully developed cases and presents itself under typical aspects in the affected regions: the scalp, as a pityriasis with abundant white scales; the hairs do not fall out; on the face, no pilary cones are seen, but a diffuse scaly redness, with tension of the skin, even ectropion, or a chalky appearance, with greasy crusts on the eyebrows and in the nasogenial grooves; on the elbows and knees, red patches are observed with thick, adherent, roughened scales, less sharply limited than those of psoriasis.

The dorsal surface of the phalanges, even more than the last-named regions is a seat of election of pityriasis pilaris; sometimes red papules agglomerated in patches are seen; in other cases only

blackish horny cones at the pilary orifices; these lesions are almost pathognomonic. The nails are striated.

The palmar and plantar regions are of a dusky red color; their horny layer is thickened, dry, fissured at the folds, sometimes desquamating; the transition into healthy skin is gradual, imperceptible.

The limbs and often also the trunk, are the seat of more or less grouped, acuminate papules and patches or thick and scaly surfaces, which may cover large stretches, almost the entire body, although a few localities at least are always exempt. The healthy spaces are angular and limited by concave curves.

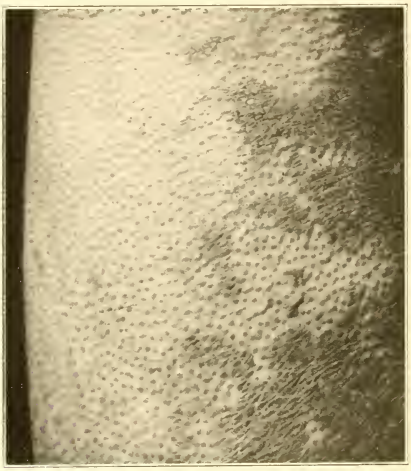


Fig. 128.—Pityriasis rubra pilaris. Anterior surface of the thigh; man aged twenty-five years; the almost generalized eruption dated back three years.

The patients sometimes complain of itching or heat and usually of a sensation of tension.

The *course* of pityriasis rubra pilaris is very variable. At the onset, the palmar and plantar regions, or the elbows and knees, or also the dorsal aspect of the phalanges and the hairy scalp may be alone affected for a period of months or years. So-called abortive cases are therefore frequently met with. Sometimes, follicular papules, grouped on the limbs or on the trunk, are the only demonstrable symptom.

As a rule, the more or less prolonged periods of invasion and confluence are interrupted by long stationary stages. Sudden extensions, as in exfoliative erythroderma, are likewise noted.

The *histological lesions* consist essentially of a laminated hyperkeratosis of the follicular infundibulum, composed of horny plugs, around a hair which is preserved or atrophied or broken off short. The granular layer persists and is sometimes even hypertrophied. The rete is thinned or slightly thickened, sometimes stretched. The congested papillary body presents a variable degree of cellular infiltration, often rather scanty and diffuse. There is nothing to suggest psoriasis or lichen.

The *nature* of the disease is shrouded in mystery. It is known to occur at any age, especially during adolescence and youth, the male sex being somewhat more frequently affected, but its etiology is entirely unknown.

In December, 1906, Milian suggested a tubercular origin of pityriasis rubra pilaris, on the ground of: (1) The remarkable frequency of tuberculosis among these patients; (2) the existence of intermediate cases between lichen scrofulosorum, true tuberculides and pityriasis rubra pilaris; (3) the positive tuberculin reaction which is obtained in cases of pityriasis pilaris. But these proofs are not sufficiently convincing. Since that time, some confirmatory cases have been observed and a goodly number of others which do not harmonize with this interpretation. It is therefore still an open question whether or not pityriasis rubra pilaris should be regarded as a perifollicular tuberculide, related to lichen scrofulosorum and akin to the pityriasis rubra of Hebra-Jadassohn.

The *diagnosis* is often very easy; in doubtful cases, confusion may possibly occur with other peripilar skin diseases, especially keratosis pilaris rubra and lichen spinulosus, or with the erythrodermas, with psoriasis and with lichen planus.

The classical *treatment* heretofore has been that of psoriasis: baths and soap, with arsenic and tonics internally. In view of the possible tubercular character of pityriasis rubra pilaris, emphasis must be placed on hygienic measures, fresh air, strengthening food, cod-liver oil. Milian was impressed with the remarkable improvement produced by injections of tuberculin. I have employed them together with injections of novarsenobenzol and have obtained encouraging but inconstant results. An attempt should also be made with radiotherapy. [I have found the *x*-ray entirely useless.]

FOLLICULAR KERATOSIS.

In the folliculoses of this kind, the inflammatory character is well-nigh obliterated; some of them may be considered as malformations.

Keratosis Pilaris Simplex.—This disease, also known as *lichen pilaris* (Bazin), *cacotrophia folliculorum* (T. Fox), *ichthyosis anserina scrofulosorum*, *xeroderma pilaris*, is extremely common. Nearly one-third of all individuals of both sexes suffer from it to some degree; it is hereditary in many otherwise healthy families.

This fact casts a doubt on the relationship of this affection to scrofula; it cannot be simply dismissed as a tuberculide. Its relations with ichthyosis, which practically always accompanies ostiofollicular keratosis, are on the contrary obvious.

Keratosis pilaris appears toward the age of two or three years, flourishes between fifteen and twenty and subsides at an adult age. Mild cases are the most common.

It usually occupies the external surface of the arms and thighs, often also the calves, the lower part of the legs, the forearms, elbows and knees, the waist and the hips, sparing fatty and moist regions.

The *symptoms* consist of dryness of the skin with roughening due to more or less marked acuminate papular elevations; these are follicular orifices filled with a very adherent grayish horny cone, in which the downy hair is rolled up as a spiral. The color of the integument is sometimes normal; in other cases, of more severe type, the follicular constituents are red or purplish, representing *keratosis pilaris rubra*.

In the course of time, the shrivelled hairs disappear, the elevations become flattened and transformed into punctiform cicatrices.

Although of the nature of deformities of embryonic origin, like the nevi, keratosis pilaris takes a course which leads to total atrophy of the affected follicles and their sebaceous gland and to alopecia of the invaded regions.

Young girls or young women frequently seek advice on account of the roughened condition and red dots on their arms. The same internal medication is recommended as for ichthyosis. Locally, it is advisable to avoid pumice stone and rubbing with alcohol, but the skin should be kept anointed with a fatty substance, a soapy salve, vaselin, or glycerol of starch containing salicylic acid. From time to time, the skin should be cleansed with green soap; [a salicylated cucerin salve is useful].

Keratosis Pilaris Rubra Atrophicans of the Face.—This affection, named *folliculitis rubra* by Wilson, *ulerythema ophryogenes* by Unna, was studied by Brocq, who pointed out its connection with keratosis pilaris simplex. It is observed in youthful or adult individuals, preferably in males.

It is situated on the eyebrows, especially at their outer third, on the lower portion of the forehead and in the parotid region; furthermore, it is not infrequently combined with simple keratosis pilaris in the elective territories of the latter. It is characterized

by a diffuse redness, with a granular surface due to the acuminate elevations of the pilary orifices, from which protrude scanty and deviated hairs. Later on, the affected surfaces become bald and the beard especially grows only very scantily; moreover, fine cicatricial spots are seen, sometimes in an anastomotic network.

The atrophic tendency is therefore more pronounced than in simple keratosis. Brocq has pointed out the kinship between this keratosis rubra pilaris of the face and moniliform aplasia; the two affections may coexist.

In pilary keratosis of the face, which is very obstinate, the condition may be improved by repeated applications of green soap; red oxide ointment is equally successful. Crossed linear scarifications may be indicated.

Lichen Spinulosus.—Under this name, first used in England, several skin diseases of unknown and probably different character have been described. Their characteristic symptom consists in the presence of more or less long and dry, filiform horny protuberances emerging from the pilo-sebaceous orifices which are themselves slightly raised and of normal or faintly pink color.

The lesion is encountered in youthful individuals and occupies diffusely the face, or the neck, or the limbs, or the buttocks (*acné corneé* of French authors); or it may be arranged as circumscribed patches on the trunk and the buttocks (acne keratique of Tenneson); or it appears in little children and covers large surfaces (lichen spinulosus of R. Crocker and Adamson).

The duration of this dermatosis is variable; several times it has been known to disappear spontaneously in a few weeks or months. In one case I have found large numbers of demodex in the affected follicles.

Analogous horny protuberances may be observed in lichen scrofulosorum, in the peripilar syphilides, etc. It must therefore be kept in mind that the spinulation is not the pathognomonic sign of a single and always identical affection.

Ichthyosis Follicularis.—This term and that of *keratosis follicularis* have been employed by various writers to designate actually non-classifiable affections which resemble either psorospermosis follicularis but without dyskeratosis, or ostiofollicular hyperkeratotic nevi, etc. A familial and contagious form has been described by Brooke.

In the *differential diagnosis* of these various follicular keratoses, it must be remembered that lichen planus may exceptionally assume the form of acuminate papules; that pityriasis rubra pilaris includes numerous incomplete, entirely localized cases; that spinulation is encountered in several diseases; finally, that the follicular syphilides are sometimes discrete and often very polymorphous.

CHAPTER XX.

TRICHOSES.

The name "trichoses" (θρίξ, τριχός = hair) is applied to the diseases and anomalies of the hairs on the head and body.

The hairs are filiform corneal structures whose root is inserted in the pilary follicles; they are a secretory product of the terminal papilla of these follicles which they cover with their enlarged extremity, known as the *bulb*. This bulb is hollow while the hair is still growing; it closes up and becomes solid when the hair has completed its development and is ready to fall out. When a hair has fallen out or been forcibly removed, unless the deep portion of the follicle has been destroyed, it is replaced as a rule by another hair, which forms in a diverticulum of the original follicle.

The structure of the hairs is very simple. They consist of more or less pigmented elongated corneal cells, forming what is known as the cortex of the hair; of an external cuticle; and of a central medullary canal, which may be absent.

Under the heading of trichoses belong: (1) the *hypertrichoses;* (2) the *hypotrichoses* or *alopecias;* (3) the *dystrophic trichoses;* (4) the *parasitic diseases* of the hairs.

Obviously, it would be logical to group the majority of the trichoses with the folliculoses. As a matter of fact, a pilary hyperproduction or an alopecia is the manifestation of a trophic disturbance of the papilla of the hair; in the tineas, the parasites attack simultaneously the root of the hair and its sheaths. But this follicular lesion is not apparent; the striking feature consists in the malformation, fall, absence or alteration of the hairs. In accordance with the plan of this book I shall therefore devote a special chapter to the trichoses.

HYPERTRICHOSES.

Hypertrichosis, an anomaly consisting in an overproduction of hairs which are larger, more abundant and more highly pigmented than is appropriate for the affected region or the age and sex of the subject—does not always represent the same type of disease. Sometimes, it accompanies congenital hyperkeratosis. The pure cases should be grouped, as already indicated by Virchow, into three classes, between which intermediate forms are encountered.

1. *Nevi pilosi* are very common, often multiple, small and lenticular, or very extensive and may cover large areas (Fig. 129, *A*) Even when they are not verrucous, the skin is usually pigmented and

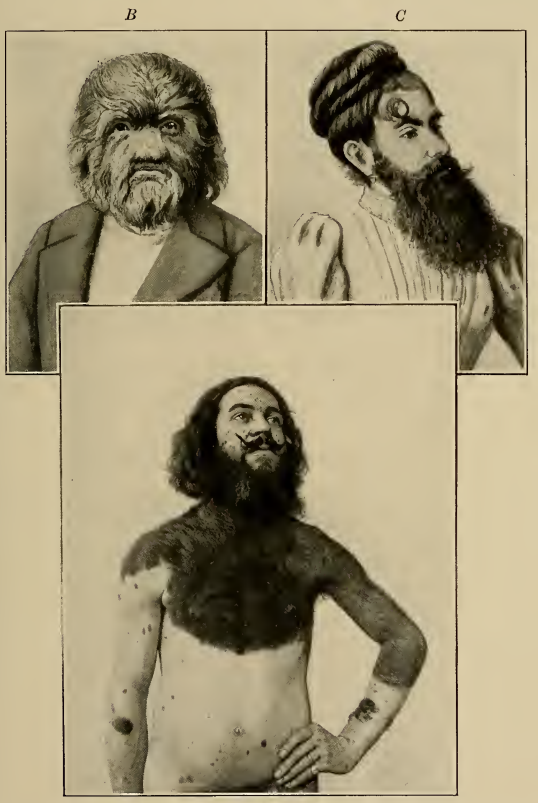


Fig. 129.—Three types of hypertrichosis. *A*, nevi pilosi; *B*, hypertrichosis fetalis; *C*, true hypertrichosis of masculine type.

nevus cells may be found in it. They are as a rule sharply circumscribed. Their arrangement is not perfectly symmetrical. They may develop after birth.

2. *Hypertrichosis fetalis*—or *lanuginous pseudohypertrichosis* of Bonnet—is seen in so-called *wild men* or *human canines* and consists in the abnormal persistence of the fetal hairs which, moreover, are hypertrophied. It is symmetrical, increases with age, occupies regions which even in persons with a very strongly developed hairy system are rather smooth, like the forehead and the nose. The hairs are wooly, soft and curly. The affected individuals usually present serious dental anomalies. Virchow designated this form as *hypertrichosis of the edentata* (Fig. 129 *B*).

3. *True hypertrichosis* must be considered separately in the two sexes.

In men it is merely an exaggeration of the normal state which is itself extremely variable. It does not really manifest itself until puberty which may be premature. Together with an abundant hairy growth of the beard and other hairy regions, an almost ape-like hairy proliferation may appear symmetrically on the chest, back and limbs; sometimes the hypertrichosis predominates in the sacral region, forming the tuft which the ancients considered as an attribute of the fauns.

Masculine hypertrichosis is not uncommonly hereditary or atavistic. It has been observed to occur not infrequently in tuberculous individuals.

In women, *hypertrichosis of the masculine type* constitutes the form which is of chief interest to the dermatologist.

The patients are usually young girls or young women who have noticed since puberty, first the development of a troublesome downy growth, followed by hypertrophied hairs on the upper lip, the chin, or the cheeks and more rarely by complete beards made up of 15 to 20 thousand hairs (Fig. 129, *C*). In some cases, the chest, the breasts, or the limbs are the seat of this abnormal hairy growth.

These hypertrichoses of young girls not unfrequently is the occasion of a real pathological obsession, known as *trichomania*, which plunges them into despair and melancholia, even in cases where the down is hardly disfiguring.

Much more common is the appearance of large hairs on the chin, or of a moustache in women of thirty to forty years or especially at the time of the menopause.

The cause of this deformity is sometimes hereditary; a natural tendency is apt to become aggravated by local irritations, the employment of depilatory pastes or of the razor and especially by epilation with the forceps. On the other hand, there undoubtedly exists a possible, but actually very inconstant relation between hypertrichosis of the masculine type and the genital functions.

This relation is indicated by cases of precocious menstruation and sexual maturity in hypertrichotic individuals on the one hand and

on the other by the hypertrichosis of the menopause. Lesser observed a girl of six years who had menstruated since the age of three, had the breasts of an adult female and was more hairy than a man of thirty years.

In both sexes, repeated local irritation, scratching, phototherapy, etc., may give rise to *regional hypertrichoses*. Other cases are of kerotic origin.

According to observations made in the course of the war, by G. and M. Villaret and others, a hypertrichosis which develops on the cutaneous territory of a traumatized nerve generally coincides with hyperidrosis and with the absence of the reaction of degeneration and indicates an incomplete injury, not a definite lesion of the nerve. Inversely, a hypotrichosis originating under the same conditions is usually accompanied by the reaction of degeneration and indicates a complete interruption of the nerve.

Treatment.—The question of treatment arises only in cases of hypertrichosis in young girls and women. At the onset, it is especially necessary to forbid all local irritations and especially epilation.

Too plainly visible downy hairs may be bleached with strong hydrogen peroxide solutions after all fat has been removed, or a peroxide cream may be prescribed. A salve made with thallium acetate (1 per cent.) will cause a large proportion of the hairs in the medicated region to drop out, but has the disadvantage of undergoing absorption and thereby acting at the same time on the hairy scalp and the eyebrows.

When the hairs are really excessively large no recourse is left but electrolytic epilation, which is radical, not particularly painful, leaves hardly visible cicatrices when properly performed but is very tedious when the hairs are at all numerous. For the technic, the reader is referred to special contributions, especially by Brocq, who has carefully investigated this question.

Radiotherapy is often demanded by these patients, but cannot at present be recommended.

Outside of the hairy scalp, its epilating action is unreliable and too dangerous to employ; in large doses, it exposes to very undesirable pigmentations and incurable spots of radiodermatitis atrophicans developing after a few years. In weak doses, a renewed and sometimes exaggerated growth may be expected after a variable length of time.

ALOPECIAS.

The term alopecia (ἀλώπηξ = *fox*) is indiscriminately applied to the fall of scalp or lanugo hairs (*defluvium*), to the resulting hairlessness and even to the congenital absence of hair.

The *congenital alopecias* [atrichia] are very rare and often familial;

in their distribution they are diffuse or regional and occasionally circumscribed. The pilary agenesia may be pure, or essential; or it may be associated with nevi, congenital hyperkeratosis, keratosis pilaris, monilethrix, more or less pronounced cutaneous atrophy, etc.

The *acquired alopecias* are divided into two classes, according to their being *diffuse* and *regional*, or on the contrary *circumscribed*. The hairy scalp is their seat of predilection and, unless otherwise indicated, the following remarks will deal particularly with this region.

Regional and Diffuse Alopecias.—Traumatic Alopecias.—Certain traumatic alopecias are diffuse. The scalp or body-hairs may have been pulled out, accidentally, with a therapeutic object or for purposes of simulation. The name *trichotillomania* designates a sort of tic or bad habit which causes some persons incessantly to pull or tear out the hairs of a given region.

Contusions and wounds give rise only to circumscribed alopecias. But the rubbing of the head on the pillow, especially at the occipital and parietal prominences in some weakly or hydrocephalic children; friction of the vertex in women, caused by combs or coiffures; friction of the clothing on the wrists or legs; scratching in consequence of pruritus—all of these induce deterioration and falling of the hairs, a condition which may be grouped under the heading of traumatic alopecia.

Pathological Alopecias.—These are much more common. It would be logical and may seem easy *a priori* to subdivide them into two groups, according as the fall of the hairs depends upon a local affection or is the result of a disturbance of the general health, such as an infectious, dyscratic, or cachexia-inducing disease.

Sometimes the local affection is evident: In this way an eczema of the hairy regions, the exfoliative erythrodermas and very extensive tineas, may lead to diffuse alopecia. I shall not dwell on this class of cases, the diagnosis of which is very easy, the treatment being that of the causative disease.

In other cases the hairy scalp appears healthy; here an investigation will disclose the existence of one of the general diseases which will be discussed further on.

In the vast majority of the cases of diffuse alopecia, however, only mild or ordinary lesions are found on the alopecic skin, such as pityriasis, seborrhea, etc., which one may hesitate to consider as causative. These conditions are interpreted by some writers as accessory and irrelevant, by others as the actual cause of the alopecia. In my opinion, these various pathological phenomena are not interdependent, but are all alike derived from kerosis.

Kerotic Alopecia and Calvities.—Kerosis is the name which I apply to an extremely frequent cutaneous dystrophy, shown by

combined manifestations such as pityriasis simplex, seborrhea, hyperidrosis and nutritional disturbances of the pilary system, in the form of hypertrichosis or alopecia. In different cases, one or several of these symptoms may predominate.

It is customary to consider as special pathological types and to describe separately, alopecia with seborrhea (*alopecia seborrheica*) with pityriasis (*alopecia pityrodes, pellicularis, furfuracea*), and the apparently essential alopecia (*alopecia senilis, prematura* and *calvities*).

However, these types are very imperfectly defined and can be included in one and the same description, a few words sufficing to point out the different clinical varieties of kerotic alopecia.

On the scalp, its seat of predilection, *kerotic alopecia* is diffuse, but regional and progressive. It begins at the vertex, at the place of the tonsure and at the sides of the forehead. As it progresses it becomes generalized and spreads at an extremely variable rate. It may stop half-way or even retrograde slightly in youthful and properly treated individuals. Often, however, it progresses inexorably, denuding the entire top of the head; it spares for a fairly long time a median island at the top of the forehead and almost always definitely the temporal and lower occipital regions, so that a semicircle of hairs is left passing from one temple to the other across the nape of the neck.

Before becoming detached, the bulb of the hairs has become solid and they will then yield to the slight traction of the brush, etc.; a few days later they fall out spontaneously.

The fall may be continuous, or it may occur paroxysmally in variable degrees. Although there are in this respect great individual variations or differences according to age, season of the year, mode of life, etc., a scalp which regularly loses from thirty to forty hairs daily may certainly be said to be in course of denudation and the ratio of the daily fall is often much higher.

The fallen hairs, which at first are healthy and of normal calibre, are always replaced; but the successive generations are more and more delicate, until they are finally represented merely by a fine down, which may disappear in its turn. The resulting condition is total baldness (*calvities hippocratica*). The skin of the scalp becomes white, smooth, glistening, polished, and seems to be atrophied or at least slightly thinned.

In the course of the development of the disease, the scalp has almost invariably become affected with the oily scales of pityriasis, with seborrhea and hyperidrosis. For a time there may have been a production of scaly crusts or more or less circumscribed and figured surfaces, accompanied by pruritus; as a matter of fact, eczematides are common on the scalp of kerotic individuals and may recur in bald persons who neglect personal cleanliness.

Varieties.—As stated above, the varieties of kerotic alopecia are very imperfectly defined.

According to authors, *alopecia seborrheica* is said to be precocious and rapid in its development, plainly regional and strongly denuding; *alopecia pityrodes*—referring to fatty pityriasis, as the dry pityriasis is not depilating (Sabouraud)—is said to be more diffuse and always incomplete; *alopecia senilis* is supposed to depend upon cutaneous atrophy and to be unrelated to seborrhea and pityriasis; it is slowly progressive and the most relentless in its advance; *alopecia prematura*, often familial, may begin toward the age of twenty years and cause baldness at twenty-five or earlier, but has such variable features that its pathogenesis is by no means agreed upon.

In women, notably in young girls and young women, an abundant fall of hairs is very common in recurrent, sometimes seasonal periods, with more or less fatty pityriasis; but it only exceptionally leads to baldness. The latter is seen in aged women and occupies the temples as well as the sinciput.

Kerotic alopecia of the beard, the moustache, the eyebrows and cilia, is much less common and accompanies as a rule fatty pityriasis or still more frequently the eczematides of these regions; it is incomplete and always temporary. Bald individuals usually have a fine full beard.

On the trunk, notably on the chest, kerosis produces on the contrary a definite loss of hair in many cases, with persistence of a few thick scattered hairs.

Etiology.—Having previously discussed the etiological factors of kerosis, I may here limit myself to stating that mental overexertion, night work, a poor dietary hygiene and in fact also a poor hygiene of the scalp, dressing the hair brush-fashion, heavy and badly ventilated skull-caps, etc., seem to predispose to progressive alopecia.

Very frequently the ordinary causes become combined with some of the pathological conditions discussed in the next section, convalescence, anemia, dyspepsia, etc., so that the differential diagnosis between kerotic alopecia and alopecia of general diseases is often extremely difficult and even impossible in combined cases. The prognosis must therefore be guarded.

[Women rarely are bald; men rarely lose their beards. In fact the same conditions affecting the scalp and the bearded region in a man may cause a complete alopecia of the scalp while scarcely thinning the beard. May we not seek the explanation of the relatively greater resistance of the man's beard and the woman's hair in an underlying biological fact? The beard of the male and the long hair of the female scalp are secondary sex characteristics and as such might be expected to possess a greater inherent resistance to injury than a useless structure like the hair on the male head.]

Treatment.—After having corrected, if practicable, all that is deficient in the hygiene and health of the patient, the kerosis must be treated systematically and for a long time with reducing agents, sulphur preparations, tar and mercurial lotions, etc. Later on, various stimulating lotions may be prescribed. By these means, the trouble is very often successfully checked, or rather the fatal outcome may be delayed.

In established baldness, I have seen a few cases of patients, who persisting in very prolonged and energetic treatment, acquired a few strong hairs on the denuded surface, but they remain thinly scattered and the result is by no means satisfactory from the esthetic point of view.

Alopecias of General Diseases.—A large number of acute infectious diseases—typhoid fever, erysipelas, pneumonia, grippe, the eruptive fevers, the erythrodermas, etc.—are followed during convalescence or after a few weeks' delay by a diffuse acute alopecia. The same may result as a sequel of childbirth, severe operations, serious traumatisms, violent emotional disturbances.

The fall of hairs in these cases may be slightly marked or so abundant that the hair comes out in handfuls and the denudation is practically complete in a few days, constituting the *defluvium capillitii* of the ancients. The alopecia may also involve the hairs of the body. The present pandemic of influenza has produced an extraordinary number of cases of alopecia. [The defluvium usually develops about two months after convalescence.]

When the scalp is not pityriasic, it is useless to cut the hairs which are left; some stimulating washes suffice and the hair grows in again as abundantly as before the disease.

Syphilitic Alopecia.—This condition may be considered to some degree as a peculiar instance of this class. It occurs very commonly from the third to fifteenth month after the infection. Its onset is often insidious. The scalp is not necessarily the seat of eruptions, crusts, etc., but there is often a combination with pityriasis.

There is sometimes a mere thinning of the hair, sometimes a *regional alopecia* [alopecia areolata] which is almost pathognomonic, with its incompletely denuded areas oscupying especially the temporal regions and the occiput. [The alopecia which is pathognomonic is what may be described as having a "moth-eaten" appearance.] The coexistence of a pigmentary syphilide of the neck is not uncommon.

This syphilitic alopecia must not be confused with pyodermic alopecia or with the alopecia areata whose patches even when numerous are usually more completely denuded and more sharply outlined. Syphilitic alopecia may also affect the hairs of the body, the beard, the eyebrows and especially the outer end of the eye-

brows. The hair always grows again, for "syphilis makes no bald heads" (A. Fournier).

In all the infectious alopecias, a paralysis of the pilary papillæ is apparently produced under the influence of toxins, comparable to that which affects the matrix of the nails under the same conditions; a very large number of hairs at once assume a solid bulb. The condition is therefore a sort of pathological moulting.

The absorption of thallium salts produces a total alopecia comparable in every way with the infectious alopecias.

Chronic diseases, anemia, diabetes, cancer, myxedema, exophthalmic goitre, mycosis and the leukemias, utero-ovarian diseases or spaying in women, depressing mental diseases, affections of the liver and intestines and still more frequently tuberculosis, give rise to chronic, diffuse, progressive, alopecias which must be traced to their true origin. In leprosy, the hairs of the face in general and those of the body fall out, whereas the hairy scalp escapes.

Circumscribed Alopecias.—The first point to be settled in dealing with a non-congenital circumscribed alopecia is to decide if it is cicatricial or not.

In a case of *cicatricial alopecia*, the surface, grain, sheen, color, consistence and sometimes the mobility of the skin are modified; the hair-follicles have entirely disappeared; atrophic, brittle, downy hairs are never present; when some hairs are left, they are of normal calibre.

Cicatricial alopecia may result from a wound, a burn, a caustic agent, or it may follow upon favus, patches of lupus erythematodes or scleroderma, ulcerative tertiary syphilides, pseudopelade, acne decalvans, etc. The denudation is permanent. The local examination and clinical history will clearly indicate the origin.

A *non-cicatricial alopecia* is primarily suggestive of alopecia areata. In the first place it is necessary to exclude traumatic alopecia or a voluntary epilation, as observed in schools and barracks, also an existing dermatosis, such as eczema, eczematide, impetigo, etc., which are easily recognizable.

It is noteworthy that impetigo, furuncle and suppurations in general, ordinarily have behind non-cicatricial, distinctly outlined, round alopecic spots, the size of a dime to that of a silver dollar, on which the re-growth of at first downy then normal hairs may be delayed for several months.

These post-impetiginous alopecias, better named *pyodermic alopecias*, due to a local blasting of the hairy territory by the toxins of the pyococci, are common in children and are often mistaken for alopecia areata. They are characterized by the macule which may be observed in their center and by the clinical data. A stimulating lotion suffices for their cure.

Alopecia areata (la pelade) [area celsi] is the most important of the circumscribed alopecia-producing dermatoses. There exists a generalized form, known as *alopecia decalvans* [or *totalis*].

Alopecia areata is characterized by smooth, sharply outlined, round or oval spots or patches of variable dimensions and number, occupying especially the scalp and the beard. These spots appear insidiously, as a rule without any particular sensation. Denudation of hair is rapid, by tufts, within a few days; it may then slowly spread eccentrically, or over a part of the periphery of the spot.

The fall of the hairs follows upon preceding imperceptible changes; they fall out with a solid bulb, many have atrophic roots; furthermore, broken atrophic, so-called alopecia hairs are found at the periphery of the patch, or even at a distance when it is about to extend.

These characteristic alopecia hairs, very abundant in some cases (*alopecias with fragile hairs*, of Besnier), very rare in the beard, are from 2 to 6 mm. long, pointed like a [wet] brush at the ends, black halfway or in the distal two-thirds, very thin, tapering and decolorized toward the root, which terminates in a slight swelling; they are accordingly club-shaped or like a note of exclamation. Being very superficially inserted, they are very easily pulled out with forceps, never breaking off.

Fresh patches are often rose-colored, slightly edematous, riddled with dilated pilary orifices containing seborrheic utricles (Sabouraud). After some time, the spot becomes depressed, ivory white, entirely smooth, soft to the touch and easily folded. This stage was called *achromatous alopecia* by Bazin.

A healing patch becomes covered with downy hairs which at first are thin, pale and very loosely implanted; these are replaced by stronger downy hairs and finally by normal hairs, actually thicker and darker than the original, but sometimes on the contrary white. The new growth may be central and centrifugal, or it may be centripetal.

The *areas* of alopecia, very variable in number, are situated at any point of the scalp, perhaps more frequently near the vertex, on the parietal and the occiput; anywhere in the beard, especially on the sides of the chin; more rarely on the eyebrows and lids, as well as on other hairy regions of the body. A symmetrical tendency has been noted in some cases and on the other hand a regional distribution in others.

As varieties may be described: a form with multiple small spots, resembling syphilitic alopecia; a form with a large patch extending like a crown from the nape of the neck, above the temples and to the forehead; this is the *ophiasis* of Celsus or *alopecia coronarius*

peculiar to children, according to Sabouraud; it is especially obstinate as are all the alopecias which affect the border of the scalp.

Alopecia decalvans is the most serious variety. It begins like the ordinary form often in very extensive areas which remain limited for several days or even several months. Suddenly, in a few days, the alopecia then becomes generalized over the entire or nearly the entire scalp, face and body, sometimes leaving a tiny tuft or a few islands of hair. In this last named form are sometimes seen a relaxation of the skin permitting it to be easily folded, *i. e., hypotonia*; and *lesions of the nails*, which will be discussed elsewhere. Slight sensory disturbances have been noted on the smooth surfaces, cryesthesia, hypoesthesias, etc.

The *course* of alopecia areata is very variable. Mild cases are cured in two to six months. Recurrences, appearance of new patches before the first have healed and relapses at any time are extremely common. There occur cases of incessant renewal and fall. Alopecia decalvans lasts from one to four years and is entirely cured in youthful individuals, incompletely in older patients.

Etiology and Nature.—Twenty years ago, alopecia areata was almost universally believed to be parasitic and contagious; at least a contagious form was admitted. The stories of epidemics in schools and barracks, quoted in support of this contention, were scattered and blown away like smoke as soon as it could be recognized by careful investigation that there was always a coincidence of sporadic cases with cicatrices and various pseudo-alopecias or with ringworm. Credit is due to Lucien Jacquet for his indefatigable work in proving the non-contagiosity of pelade; his thousands of experimental inoculations on himself and on already affected, therefore predisposed, subjects, did not yield a single positive result.

In favor of a nervous origin of alopecia areata have been quoted the experiments of Max Joseph who through division of the occipital nerves in cats produced bald spots which, as a matter of fact, had only an apparent analogy with alopecia areata; and on the other hand, the not uncommon coincidence of alopecia areata with neuralgias, headaches and an "unbalanced" nervous state.

In the dystrophic theory of Jacquet, the predisposing role was attributed either to a complex organic deterioration, as indicated by urinary analysis, or to hereditary factors. On the soil thus prepared, the alopecia was assumed to be elicited and focussed through local peripheral, or visceral, or central irritations. Among the irritations starting the reflex, those of dental origin are the most common; very often the eruption of the teeth, more particularly the wisdom teeth, or dental caries, alveolar inflammations, badly fitting dentures and so forth can be held responsible. There is even a certain relation between the localization of the bald areas and that of the irritative focus.

Taking up the question *ab ovo*, Sabouraud and his school started of recent years a new inquiry concerning the etiology of pelade. It appears that this affection is hereditary or familial in at least 22 per cent. of the cases; the affection is only half as frequent in the female as in the male sex; its maximal frequency of onset is, in both sexes, between the age of six and twelve years; it is furthermore observed with relative frequency in women at the time of the menopause or after a prolonged suppression of menstruation, through spaying, for example, and more rarely in the course of pregnancy.

Statistics finally show a connection between alopecia areata and thyroid disturbances, especially exophthalmic goitre, where it is apt to be chronic and severe. As to the etiological relations with acquired or congenital syphilis, these are neither clear nor direct; syphilis could be demonstrated in only about 10 per cent. of the cases of alopecia areata.

These data are undoubtedly interesting, but on the whole it must be admitted that the real nature of alopecia areata still remains unknown.

Treatment.—Since it is known that alopecia areata is never and in no degree contagious, the *prophylaxis* of this affection has assumed an altogether different direction. There is now no reason to isolate the patients or to keep them away from gatherings, schools, workshops, barracks, etc., to refuse them a clean bill of health, nor to suspect hairdressers or barbers, caps, hats, pillows, etc., of having transmitted a disease which is non-transmissible.

The treatment of alopecia areata and the prophylaxis of relapses or recurrences, are therefore purely individual.

An effort must be made in a given case to determine the existing factors of nervous and trophic loss of balance, in order to treat its general and deep causes. In the presence of overstrain, bad hygienic conditions, organic or functional disturbances, it may be necessary to recommend relative rest, life in the open air, in the country or the mountains; dry or alcoholic friction of the entire body; hydrotherapy in its tonic or sedative forms, or perhaps various opotherapeutic measures, or the reconstructives, phosphates, arsenic, etc.

On the other hand it is very important to look for the localizing causes, as it were. A bad condition of the teeth and gums requires attention, even in the absence of pain, congestion, neuralgia, etc. This point must be emphasized; I have personally seen, like Jacquet, a considerable number of cases which had resisted all local medication heal of their own accord with suggestive rapidity after expert treatment at the hands of a dentist. Sometimes the "peladogenic" focus is to be found in the ears, nose or pharynx and one must proceed accordingly.

The *local measures* may be summarized as follows: repeated, sufficiently energetic but not exaggerated stimulation of the denuded areas. This stimulation may be mechanical, being obtained by massage, pétrissage, brushing, twice daily or oftener; in the stage of re-growth, it must be remembered that of all stimulants epilation acts the most directly upon the pilary papillæ. Chemical stimulation may be accomplished by means of iodized, acetic or ammoniacal, chloroform or alcoholic, or other mixtures selected from the list of stimulating and rubefacient lotions; the applications should be made every day or at longer intervals. [Pure liquified phenol allowed to act till the surface begins to whiten, then de-ionized by means of alcohol, applied once in two or three weeks, is an excellent local stimulant.] Vesication or blistering, more particularly by means of a liquid vesicant painted on with a brush, may serve to whip up the pilary growths, as it were. Physical stimulants may also be employed, such as faradization, high frequency currents, etc. I have witnessed in my laboratory, in cases of alopecia decalvans, a remarkable re-growth under the influence of radiotherapy, strictly limited to the stimulated regions; of course the *x*-rays must be employed in lower dosages than those which cause epilation (2 to 3 units H every fifteen or twenty days). Numerous authors have recently recommended the employment of ultra-violet rays.

Salves and plasters which are sometimes given to these patients possess no special virtues.

The condition of the scalp should be attended to as a whole, treating pityriasis, seborrhea, or concomitant lesions if present.

In order to facilitate the local treatment in cases with extensive or numerous areas, it is advisable to cut off or shave the remaining hair. In this case a wig will have to be worn, if the patient desires to conceal his condition. When the patches are not very extensive, they can be hidden more or less effectually by touching them up with burned cork.

DYSTROPHIC TRICHOSES.

Leukotrichia and Canities.—Congenital absence of pigment in the hairs is known as *leukotrichia*. It is generalized and accompanied by a lanugo-like atrophy in albinism, which is very rare, or it may be partial, limited to a tuft of hair, as occurs hereditarily in certain families.

Canities is an acquired achromia; the hairs on the scalp and body turn grayish, then gray and finally entirely white. It is *physiological* after a certain age, but very variable according to races, families, individuals and mode of life; so that in a given case it can be designated as *senile* or as *premature*. Its distribution and relative development on the scalp, the beard and the other hairy regions are so vari-

able that no summary account is possible and all that can be said is that the condition is essentially diffuse and progressive.

Pathological canities, more or less extensive and diffuse, is seen in the course of several nervous diseases and of cachexia.

Numerous cases have been quoted in which canities is said to have developed suddenly, in a single night, for instance, under the influence of extreme terror; Dr. Parry is said to have seen the hairs of a sepoy who had been tied to the muzzle of a cannon whiten in half an hour. This is highly improbable. I do not know of any observation on sudden canities in the course of the last four years, which have certainly been prolific of unspeakable horrors.

Sometimes, a partial canities which may be temporary, follows after baldness due to alopecia areata, erysipelas, and so forth.

The mechanism of the decoloration is unknown. The hairs very rarely whiten, gradually beginning at the root or at the free end. Cases of *ringed canities* [pili annulati] have been reported, with alternately white and colored segments. As a rule, the blanching is total, progressive and more or less rapid for a given hair.

The penetration of air between the cells of the hairs does not sufficiently explain the whiteness; there is not only failure of production but actual *destruction* of the pigment, either through special pigmentophagous phagocytes, as claimed by Metchnikoff, or more probably through consumption of the pigment *in situ.*

The *treatment* of canities is practically illusory. The role of internal medication and hygiene is limited to raising the general nutritional standard. Stimulating applications, heating the scalp according to some, or epilation of the first white hairs may at the onset delay the appearance of canities.

Actually, the only corrective agent which certain individuals are obliged to employ for business reasons, consists in the use of *hair-dyes.* With the exception of burned cork, or henna which gives a red or blond color or a brownish tint, according to its mode of application and hydrogen peroxide which reddens and bleaches the still pigmented hairs, all dyes may be injurious; several are positively dangerous, more particularly those containing paraphenylendiamin. The least harmful are perhaps the silver nitrate and pyrogallic acid preparations. Numerous formulas may be found in special works on this subject.

Trichorrhexis Nodosa.—Under this name is designated a very common affection of the beard in men and of the scalp and pubic hairs in women, characterized by a localized splitting of the hair, the fibers becoming separated in the form of two brooms pushed into each other.

This results in the appearance of white nodosities, at the level of which the hair bends and is easily broken off. These thickenings are

numerous, especially toward the free end and lead to shortening of the scalp or body hairs.

This affection was considered for a long time as parasitic and contagious. Sabouraud, who found it in nearly all the old shaving brushes examined by him, showed it to be traumatic and due to an excessive withdrawal of oil through abuse of soapy washes and lotions. The remedy is therefore to cut the hairs below the fractures and to keep them constantly oiled.

Trichoptilosis.—Trichoptilosis is a cleavage in the longitudinal direction of the hairs on the scalp or of long beard-hairs, which become forked at their extremity. Severe chronic diseases or constitutional weakness may perhaps act as predisposing factors although it is also observed in perfectly healthy individuals. The disease seems to depend upon an exaggerated dryness of the hair. The treatment is that of trichorrhexis.

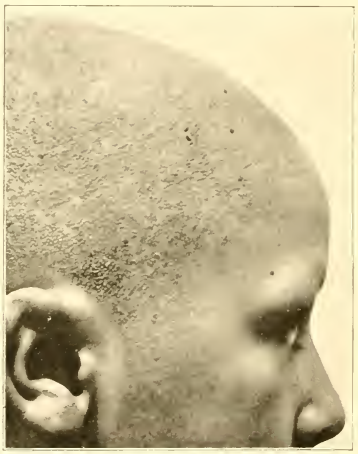


Fig. 130.—Monilethrix, showing the scalp and the parotid and palpebral regions in a child aged nine years.

Monilethrix or Aplasia Moniliformis.—This is a rare, familial and hereditary congenital dystrophy, related to keratosis pilaris and ichthyosis (Fig. 130).

It consists of a peculiar conformation of the hairs which are alternately and regularly constricted and swollen, spindle-shaped, dry, shrivelled, brittle and usually very short; the fractures occur at the

level of the constrictions; the thicker portions are more pigmented (Fig. 131).

One is inclined to admit that the formative hair-papilla undergoes changes of dilatation and atrophy, comparable to a pluridiurnal rhythmic pulsation. The follicles usually present the lesions of keratosis pilaris and often form an acuminate elevation which may ultimately become replaced by a minute cicatrix. The scalp is especially affected and generally appears entirely denuded. Hallopeau showed that the pilary system of the entire body may be involved. Sometimes this dystrophy slightly subsides with advancing age.

The treatment is that of keratosis pilaris.

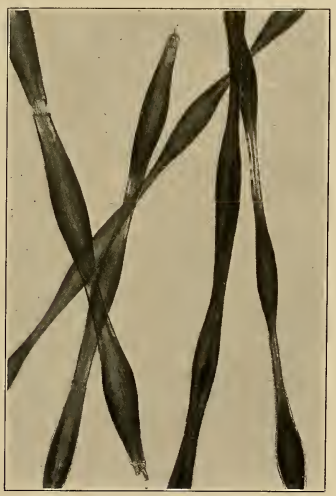


FIG. 131.—Monilethrix.

PARASITIC TRICHOSES.

There are two kinds of parasitic diseases of the scalp- and body-hairs: (1) the *tineas,* in which the parasites reach the hair at its root and invade the follicle as well as the surface-epidermis; (2) the *trichomycosis,* in which only the shaft of the hairs is affected.

Tineas.—The name of tineas must be reserved at the present day for a group of parasitic dermatoses of the hairy scalp due to fungi. They are extremely interesting not only for dermatologists

but for all physicians, representing very insidious diseases, sometimes hard to recognize, of proverbial tenacity, difficult to treat and, moreover, highly contagious. They have, accordingly, a social importance.

The tineas are three in number: *Tinea favosa* (favus), *tinea microsporica* and *tinea trichophytica*. The last two may be combined under the name of *tinea tonsurans*. The general discussion of their parasites will be found in the chapter on parasitic dermatoses (Chapter XXV).

Children alone are liable to contract tineas, the reason for this privilege being unknown; it must be assumed that in them the chemical constitution of the scalp differs from what it will be after puberty. Aside from the matter of age, there exist no conditions of immunity. All tineas result from contagion, either direct by contact or more generally indirect by mediation of toilet articles, combs, brushes, clippers, scissors, towels, hats, etc. Transmission between children of the same family is extremely common. Real epidemics may be observed in schools or gatherings of children where an unrecognized tinea-carrier has been admitted.

With these remarks on the general etiology, each tinea will now be discussed separately.

Tinea Favosa [Favus].—The hairy scalp is the seat of predilection of favus. At the onset, the achorion vegetates on the surface, only in the horny layer, where it causes red and scaly spots; this stage usually passes undetected.

In the fully developed stage, the most typical clinical form is that known as *favus scutularis*, the "favus à godets" (favus with cups); it is described as *favus urceolaris* when the scutula are discrete, regular, centered by a hair and of a bright sulphur-yellow color; *favus squarrosus*, when they are confluent, misshapen and conglomerated in grayish, powdery, scaly crusts held together by dried pus.

The spots or patches of favus are of very variable extent, sometimes larger than the palm of the hand. The entire scalp may be invaded except a border about 1 cm. in width which curiously enough always remains free.

On removing the cups (p. 516) with a curette, there is found below either a smooth depression or a suppurating ulcer or at any rate a subacute dermic inflammation with a tendency to cicatrization. In long-standing patches of favus, cups and crusts are accordingly seen intermingled with smooth, pinkish cicatrices of irregular shape.

On the favus patches the hairs have in part disappeared; those which persist emerge in tufts from the interstices of the crusts. They are dull, discolored, resembling tow. They do not break easily but yield readily to traction, with their root surrounded by its swollen,

moist, white or hyaline epithelial sheath. The microscope readily reveals the mycelium in the hairs at least in the first portion of their length.

The hairs which have fallen out are not replaced. Favus of the scalp leads more or less rapidly to cicatricial alopecia, arranged in spots or networks, characterized by its smooth, shining, more or less reddened and sharply outlined surface; here and there, crinkled hairs of normal thickness and length persist isolated or in small groups.

In other cases the cups are not apparent and it is the dull and grayish appearance of the hairs emerging from the affected regions which indicates the condition present.

The *pityriasic form* of favus manifests itself as distinctly outlined spots, covered with dry gray scales; the microscopic examination of the scanty and lustreless hairs establishes the differential diagnosis from psoriasis or eczema.

In the *impetiginous form* of favus, crusts especially are seen, gluing the hairs together and affording a lodging for pediculi. There is always a history of the lesion dating back several years.

Removal of the crusts exposes a pinkish bald surface; after some time, scutula appear, pierced by lustreless hairs infiltrated with mycelium.

The *alopecic form* very closely suggests pseudo-favic alopecia. There are no crusts or scales, but spots or islands of cicatricial alopecia; at their circumference are seen slightly papular folliculitides; a microscopic examination of the emerging hairs is necessary to establish the differential diagnosis from acne decalvans.

Tinea Microsporica.—Tinea tonsurans with small spores—or tinea of Gruby-Sabouraud—is frequent in children from four to ten years, especially in boys; it is extremely contagious. When left untreated, it disappears spontaneously at about the age of fifteen years.

It may be recognized from a distance as round or oval, large or medium-sized, distinctly outlined patches of a dusty appearance, covered with gray foliated scales, whence emerge only a few healthy hairs nearly all being broken off short, of a length of about 3 to 5 mm., lustreless and of an ashen gray color, all inclined in the same direction (Fig. 132).

It is seldom that only a single patch is present; generally from four to ten can be counted.

The most recent patches are lenticular or nummular in size; the oldest may measure 5 to 6 cm. in diameter or more. The condition has therefore been described as having large patches but small spores. Diseased hairs scattered outside of the patches can never be demonstrated. But the hairy scalp may be involved as a whole.

On grasping the hairs of a patch of microsporic tinea between the nails of the thumb and index finger, or between the blades of a pair of forceps, a certain number can be easily pulled out without pain, for they break off a short distance below the epidermic surface.

Viewed with a lens these hairs are seen to be surrounded by a dull white sheath for a distance of 4 or 5 mm. Microscopical examination, after the action of 40 per cent. caustic potash solution, shows this sheath to be formed by a thick and regular layer of round or polyhedral, somewhat uneven spores, measuring from

Fig. 132.—Tinea tonsurans microsporica. The cultures yielded microsporon lanosum.

2 to 4 μ in diameter; the hair thus resembles a rod dipped in glue and then rolled in fine sand. The spores do not generally seem to be accompanied by mycelic threads, are not arranged in short chains and are evidently ectothrix [outside the hair-shaft] (Fig. 133).

Careful inspection of the hair after it has been freed from its sheath of mosaic-like spores, may nevertheless reveal delicate mycelic filaments, with widely separated septa, dividing from above downward dichotonously, with superficially emerging branches, which perhaps give rise to the spores. At the lower portion of the microsporic hair broken off near its bulb, numerous mycelic threads can sometimes be seen, constituting what is known as "Adamson's fringe."

The presence of friable hairs clinically differentiates the small-

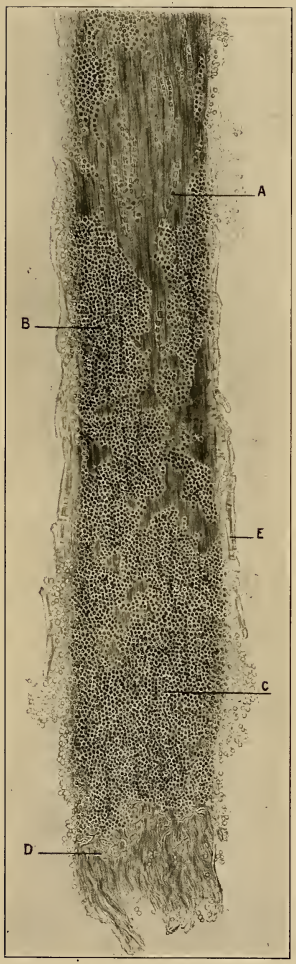


FIG. 133.—Radicular portion of a hair in tinea microsporica (microsporon lanosum.) *B* and *C*, microsporon sheath; at *A* the subjacent threads of mycelium are shown; *D*, mycelic fringe of Adamson; *E*, epidermic cells of the cuticle or of the follicle. After Sabouraud. × 260.

spored tinea tonsurans from pityriasis capitis, which moreover is generally diffuse; from psoriasis of the scalp and from the form of dry eczema named *tinea amiantacea* by Alibert. In these various affections, on the contrary, the length of the hairs is preserved, they are solid and fall out as a whole without breaking. The differences between the small-spored tinea tonsurans and trichophytosis of the hairy scalp will appear from the following:

Tinea Trichophytica.—Trichophytic tinea tonsurans—or tinea *with large spores*—likewise affects children exclusively; but it may be prolonged until about the twentieth year or exceptionally later. It is actually twice as common in Paris as the small-spored tinea, especially among girls. In contradistinction to the latter, it manifests itself as small patches, scattered in large numbers, or rather as numerous dots, each made up of a few diseased hairs; these dots become fused and may form larger patches, of any shape, but a large number of healthy hairs which hide the diseased hairs usually persist. This tinea has therefore much less striking symptoms and may escape an inexperienced observer.

A distinction must be made between two principal varieties, two clinical types, depending upon different trichophytic species. Their distinctive features are as follows, according to the description of Sabouraud:

1. The diseased hairs, intermingled with numerous healthy long hairs, may be gray, broken off at a height of 2 to 4 mm., bent in all directions, bristling as it were; the epidermic surface is covered with dry or greasy, rather thick scales, containing twisted infected hairs; this form constitutes *trichophyton* with *crateriform growth* [in cultures; briefly T. crateriforme].

2. In the other variety, the diseased patches between the surviving hairs are dotted with black points resembling powder grains and sometimes with follicular elevations analogous to those of keratosis pilaris; no diseased hairs protrude; they are enclosed in the horny layer, broken off flush with the surface or twisted on themselves in the follicular ostium. These are cases of *trichophyton* with *acuminate growth* [T. acuminatum].

The hairs for microscopic examination must not be taken haphazard or pulled out with the fingers, for the tinea would then remain unrecognized. Remnants of hairs must be looked for and extracted with fine pincers or with a needle. These diseased hairs will be found to be packed with spores larger than those of microsporon and lodged in the substance of the hair itself; these trichophytons therefore are endothrix (Fig. 134).

If the spores are square, lie in tiers or bands with resistant [coherent] mycelium, the case is one of trichophyton crateriforme. If the spores are rounded or oval, easily separated, the myecelium

brittle and the hair resembles a bag of nuts, the case is one of trichophyton acuminatum.

According to the investigations of Sabouraud (1908), this microscopical difference is not absolute, however, and cultures are required for the differentiation of the two species.

Tinea trichophytica is more often overlooked than confused with other affections. Not infrequently it is a patch of *herpes circinatus* occurring [on the glabrous surface] on the little patient or some one in his invironment that first attracts attention; or there may be a diffuse alopecia accompanied by a slight scaling on the scalp.

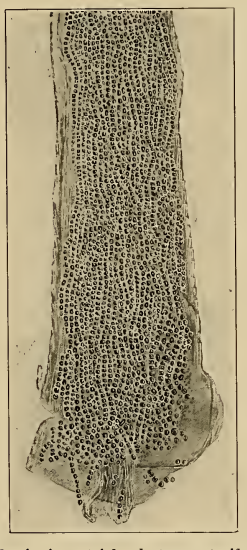


Fig. 134.—Hair in trichophytic tinea, trichophyton crateriformis. Threads composed of quadrangular segments forming bands of mycelium. After Sabouraud. × 260.

Every pityriasis with multiple spots and all *dry eczemas* localized on the scalp of children, require a minute and painstaking search for hairs broken off or enclosed in the epidermis and such will very often be found provided one knows how to look for them.

It is superfluous to emphasize that the broken hairs of alopecia areata which are dry, clubbed, thinned at their base and can be extracted without breaking, in no way resemble the hairs in tinea.

Treatment of the Tineas.—No *general treatment* is required, but these patients recover more promptly when their general condition is improved and when they are placed under excellent hygienic conditions, in the country, at the seaside, etc.

The *prophylaxis* necessitates immediate strict isolation of the patient, especially from other children.

Favus-carriers are dangerous for everybody; children suffering from the microsporic and trichophytic forms can transmit to adults only a readily curable herpes circinatus. All tinea patients, even when collected in a special school reserved for them, like the school of the St. Louis Hospital in Paris, must keep the head constantly covered and be regularly cared for.

In treating a case of tinea, it is indispensable to begin by cutting the hair short with scissors, repeating this every eight or ten days. The next indications are to outline all the diseased points, to remove or destroy the parasites by appropriate measures and to preserve the healthy portions.

In a case of tinea tonsurans, after the scalp has been washed with soap, it is advantageous to paint it all over with tincture of iodin diluted with three parts of alcohol; this serves to bring out the affected regions and protects the healthy parts from auto-infection. These soapy washes and iodin applications may be repeated every day or every other day.

Where a radiotherapeutic apparatus is available, epilation by the *x*-rays which evacuates the entire contents of the follicles is the method of choice. As shown by Sabouraud, tineas are curable in a relatively short time by this method—from four to six months, for example, instead of two or three years consumed in the old methods. All the spots are successively exposed to the rays, administering the necessary doses, namely 5 units H. in one session. When the spots are very numerous and scattered, it is necessary to treat the entire scalp, which requires twelve applications, in one or two days. Great care must be taken in outlining the exposed territories, so that no strip of skin either escapes or receives a double dose. The employment of the method therefore requires a perfect apparatus, wide experience and great care. Between the fifteenth and twentieth day, all the hairs together with their roots are cast off; by the thirtieth day, neither hairs nor parasites are left and the child is no longer contagious. The re-growth of hair begins at the end of two and a half months and is complete five months after the session; it goes without saying that during this period the children must be carefully watched. [It is advisable to paint the scalp with the dilute tincture of iodin once a week for several weeks after the raying.]

When radiotherapy is not available, a strip 8 mm. in width must be epilated with forceps around each patch, in order to estab-

lish a safety zone; and then, as well as possible, the patches themselves, where the hairs are unfortunately brittle; these epilations to be repeated every ten or fifteen days. Furthermore the spots should be painted every day with iodin tincture diluted to one-fourth strength; they should be dressed with iodized vaselin or with a chrysarobin salve; occlusion should be maintained by means of zinc gelatin or strips of adhesive plaster. It has also been recommended to rub the spots every fifteen days with a pencil of croton oil contained in cocoa-butter. The last named topical agent gives rise to folliculitis with expulsion of the infected hairs, but is liable to leave cicatrices; its action must be closely watched and the inflammation quieted with moist dressings. The treatment is continued in this way until a cure seems to have been accomplished.

Without radiotherapy, a properly treated small-spore tinea tonsurans lasts about eighteen months; many cases are prolonged for two or four years. This tinea finally always gets well without cicatrices, unless these have been caused by the treatment.

The prognosis of trichophytic tinea is aggravated by the multiplicity of the affected points and the difficulty of discovering them; moreover, it does not disappear spontaneously until the age of eighteen to twenty years. By means of correct treatment, a cure can be obtained in less than one year. It often happens, especially in the trichophytoses, that two or three follicles resist almost indefinitely; one is justified in destroying these by electrolysis, by the thermocautery, or by the application of a small droplet of croton oil, introduced with a needle. Many microscopical examinations, repeated every month and yielding negative results, are necessary before the case can be pronounced cured.

In tinea favosa the treatment begins with the removal of the crusts and cups by means of moist dressings, salicylated vaselin, or the wearing of a rubber cap, together with frequent shampooing. When the scalp is clean, epilation is performed either by means of the *x*-rays or with forceps, which is more efficient in these cases; this is repeated every month. In the interval, the parts are painted daily with dilute iodin tincture or carbolized glycerin. Various ointments of sulphur, copper sulphate, mercurial salts, etc., have also been recommended, but are of no advantage.

The treatment of favus by epilation, formulated by Bazin, requires at least six or eight months, sometimes over a year. A cure can be considered as probable only after no cup and no infected hair has reappeared for three months after the last epilation. As a recurrence is even then not excluded, it is necessary to watch the patient during at least another six months.

TRICHOMYCOSES.

These are parasitical affections of the hairs, affecting their free shaft, but not the root or the follicle. Several varieties are known:

Trichomycosis Vulgaris (Lepothrix of Wilson, or Trichomycosis Palmellina (Pick) is common in all countries, in the axillary and genital regions, in persons who neglect the demands of cleanliness. The hairs become dull, roughened, nodular, assume a yellowish or reddish color, but are not brittle; there is often a regional hyperidrosis and chromidrosis.

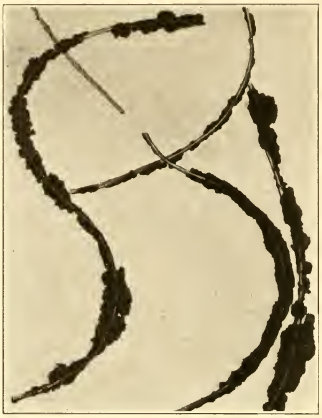


Fig. 135.—Trichomycosis vulgaris; axillary hairs under the microscope.

Under the microscope, very adherent granular concretions are seen on the hairs, forming an irregular sheath, a sort of *rugous cortex* (Fig. 135) or nodules at intervals. They consist of zoöglea of cocci, attached to erosions of the cuticle of the hairs and agglomerated in a very hard layer. Colombini successfully cultivated and inoculated this parasite.

There exist tropical varieties, such as that studied in Ceylon by Castellani (1912), due to *Nocardia tenuis*.

Washing with soap and antiseptic lotions or ointments suffices for the treatment; shaving of the hairs is only exceptionally required.

Piedra (Trichosporosis Nodularis Tropicalis) gives rise to nodes in the hairs, sometimes of the beard, arranged without order and conveying a sensation of roughness on touch. The rounded or spindle-shaped nodules, sometimes of the form of a unilateral shell,

are whitish, extremely hard and adherent (Fig. 136); they do not make the hairs brittle; Juhel-Renoy counted 23 on a single hair 60 cm. in length.

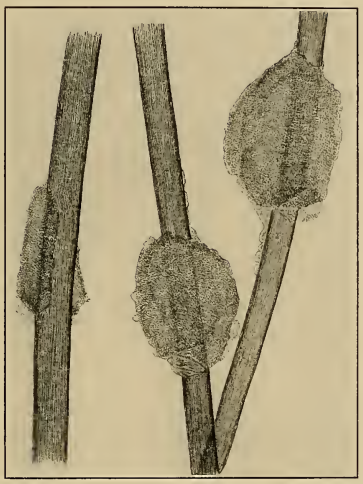


Fig. 136.—Piedra nodules completely or partially surrounding the hair.

They are composed of fairly large spores, belonging to one or various species of trichosporum, heaped up and agglutinated under the cuticle of the hair. This affection is observed in South America, especially in Colombia, in the Balkan Peninsula and exceptionally in temperate climates.

CHAPTER XXI.

ONYCHOSES.

The *nail* is a horny plate resulting from a special type of keratinization. This particular mode of keratinization is limited in man to the floor of a deep depression in the epidermis of the dorsal aspect of the terminal phalanges.

The ungual plate or *nail-plate* is of convex shape, especially in the transverse direction and its *root* is inserted into this depression, which has the form of a groove or notch. The root is bevelled on its lower surface, the bevelled edge corresponding to that portion of the invaginated epidermis which secretes the nail and which is known as the matrix of the nail (Fig. 137).

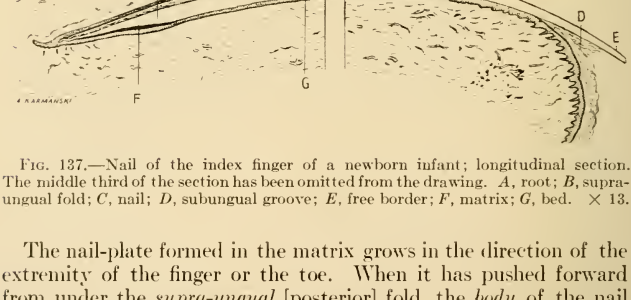


Fig. 137.—Nail of the index finger of a newborn infant; longitudinal section. The middle third of the section has been omitted from the drawing. *A*, root; *B*, supra-ungual fold; *C*, nail; *D*, subungual groove; *E*, free border; *F*, matrix; *G*, bed. × 13.

The nail-plate formed in the matrix grows in the direction of the extremity of the finger or the toe. When it has pushed forward from under the *supra-ungual* [posterior] fold, the *body* of the nail rests on the *bed* of the nail and its borders are insinuated under the *lateral folds*. Its extremity is free and passes above the *subungual groove*.

The growth of the nail, which is about 3 mm. monthly, therefore depends upon the activity of the ungual matrix. Any lesion of this matrix as a whole will manifest itself in an arrest of growth and may be finally marked by a transverse ridge on the ungual plate when this lesion has been temporary; by an atrophy of the nail when it is

permanent. The partial lesions of the matrix will result in a spot when they are temporary, a streak or a longitudinal band when they are persistent. The thickness of the nail seems to depend upon the degree of slanting of the matrix.

The bed of the nail enters to only a very slight extent into the formation of the nail which is not thicker at its free extremity than at its root. Nevertheless pathological changes of the nail-bed play a certain role in the onychoses.

The diseases of the nails, known as *onychias* or *onychoses*, therefore depend on pathological changes of the ungual matrix and to a small extent on those of the nail-bed; their inflammatory forms are designated as *onyxis*.

It is customary to describe with these onyxis the *perionyxis*, namely inflammations of the supra-ungual and lateral folds which are clinically hardly separable.

In a general way, the onychoses are very common. They may depend on (1) *Congenital malformations;* (2) *local causes*, various traumatisms or parasitic affections (onychomycoses); (3) a *localization* of *various dermatoses;* (4) the effect of a *general disease;* (5) *trophoneuroses*.

These very different *causes* and perhaps also still others of unknown nature, give rise to equally variable lesions. It must be emphasized, however, that no absolute conformity exists between a given cause and its apparent effect upon the nail; identical or related causes may produce very different symptomatic pictures, while on the contrary the same appearance may result from a variety of causes.

On account of the difficulty of biopsies and the rarity of post-mortem examinations, the *pathological anatomy* of the onychoses is very imperfectly understood.

The *diagnosis* of the onychoses is very often difficult and may remain open, unless the lesion is a rare and characteristic one, or the nature of the affection is elucidated by the presence of other cutaneous localizations. In spite of the remarkable work of Heller and numerous special contributions, this subject still remains somewhat obscure.

Congenital Malformations.—These may consist, on both extremities, in the complete absence of one or several nails (*anonychia*), sometimes the nails are replaced by a horny mass (*epidermic nail*); or they may be found to be atrophic, thin, concave, shovel-shaped (*koilonychia*); or they may be hypertrophied (*scleronychia*), thick, blackish, roughened, curved transversely, claw-like (*onychogryphosis*), or flakey or longitudinally striated, or wavy, etc. These malformations are not uncommonly familial, transmitted through several generations, sometimes associated with malformations of the

fingers, dystrophies of the hairs or other hereditary errors of conformation, as well as with various keratoses. It must be kept in mind that malformations of hereditary origin like the nevi sometimes do not appear until advanced childhood or still later.

Traumatic Onychoses.—Wounds, ecchymoses, avulsions of the nails, subungual foreign bodies, traumatic onyxis and perionyxis, ingrown nail, etc., belong to the domain of surgery.

Onychophagia, a bad and sometimes unconscious habit of gnawing the nails, is usually connected with other stigmata of degeneration or with nervous disturbances. It is observed in children of both sexes and even in adults. It is partly referable to heredity, but also largely to imitation; in some schools, more than one-third of the children bite their nails.

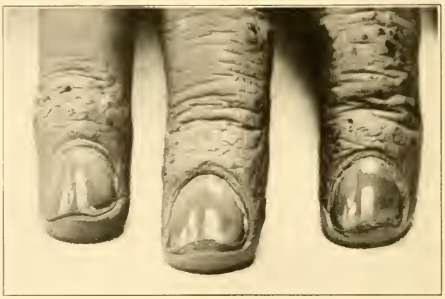


Fig. 138.—Finger nails worn down by scratching. From a case of prurigo vulgaris with eczematization.

The gnawed nails no longer have a free border; in the extreme degree, they are reduced to transverse stumps, the pulp of the fingers rising up as a cushion in front. This unclean habit may become dangerous through the absorption of pathogenic germs which have lodged under the nails. Compulsory measures and physical agents are rarely efficient; psychotherapeutic treatment is preferable.

Worn-off nails, combined or not with various artificial colorations, are encountered in a large number of manual occupations.

In chronic pruritus and the prurigos, the free border of the nails may become concave and worn, their surface being as smooth as a mirror (Fig. 138); this appearance indicates habitual scratching to the dermatologist, even when the patient is unaware of the habit or wishes to conceal it.

The popular name of *hang nails* is applied to small traumatic

tears or epidermal elevations of the posterior fold and the lateral folds; they easily become the source of infections, lymphangitis, etc., and should be carefully trimmed [sterilized with iodin] and covered with collodion.

Onychomycoses.—The ungual lesions produced by the achorion and the trichophytons (Chapter XXV) differ only by mere shades. The diagnosis is based upon the possible coincidence of other localizations of favus or trichophytosis, and especially on the microscopical examination.

Onychomycosis favosa is rare and, as a rule, secondary to favus of the scalp or body which may, however, have healed while the ungual lesion still persists. It begins in the subungual grooves or at the lateral borders, as straw-colored yellow spots which are seen shimmering through the nail-plate; these spots grow, raise the nail, which finally becomes cloudy, thickened, fissured, locally puffed up, loosened and exfoliates. At last nothing is left of it but roughened and striated débris. The growth of the achorion under the nail gives rise to a horny substance (Truffi); the nail plate itself is invaded secondarily; but according to Pellizari, it may be attacked primarily. As a rule, the majority of the finger-nails are involved; very exceptionally the toe-nails.

The *onychomycosis trichophytica* is generally caused by trichophytons of animal origin. It is more frequent in adults than in children and often coincides with trichophytosis of the beard or of the hairless skin. Several nails are generally involved, without regard to order. The lesions begin under the free border or under the lateral borders of the nail, as grayish spots with irregular or vague borders, not so yellow as in favus. A beginning at the root also is said to have been noted. When the nail itself is invaded, its external table may be preserved or not; in the former case, the nail is greatly thickened, opaque, striated like rush-pith, friable at its free border, concave, or on the contrary curved in and claw-shaped; in other cases the external table is fissured and worn and the nail eroded, spongy, roughened, dirty and more or less destroyed.

The onychomycoses are painless. They may undergo a spontaneous cure, but when left untreated usually last a very long time, up to twenty years or more; they are, moreover, very rebellious to treatment.

In a general way, oxychomycoses should be suspected in the presence of any chronic onychosis of obscure, supposedly trophic cause, etc.

Microscopical examination of nail-dust, scraped off and heated on a glass slide in a 40 per cent. potash solution, shows mycelic elements, which are shorter and more irregular in favus than in

trichophytosis; but the distinction is not easy. Cultures of ungual trichophytons often fail for no known reason.

The medical *treatment* of the onychomycoses consists in scraping as much as possible from the thickness of the nail and in having the patient wear every night for many months a dressing of cotton soaked in Lugol's iodin-iodide solution, covered with a rubber finger-stall which does not compress the finger. Various salves or ointments containing reducing or antiseptic agents may also be employed.

Surgical treatment is more rapid, consisting in avulsion of the nails after local anesthesia; iodin-iodide dressings will prevent re-infection of the new nail.

Onychoses of the Dermatoses.—There exists a *pyococcic onyxis*, generally due to an association of staphylococci and streptococci; it might also be designated as *impetiginous onyxis* (Fig. 139).

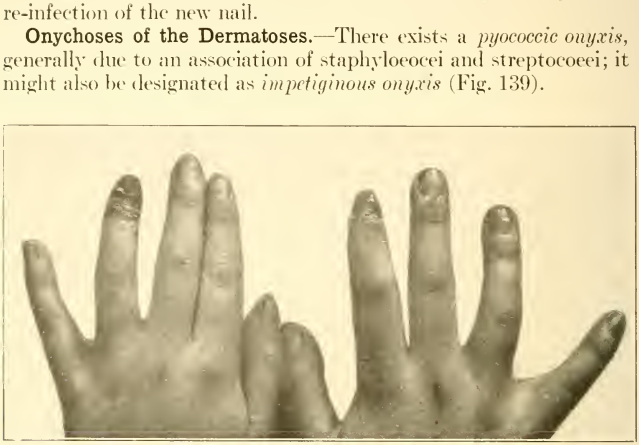


FIG. 139.—Pyococcic onyxis and perionyxis of the right index finger and the left ring finger, of one year's standing, in a girl aged ten years.

Although attention has been called to it, notably by Sabouraud, it is not sufficiently known and is a frequent occasion of diagnostic and prognostic errors. It is encountered especially in children and youthful individuals, but also in adults, on one or several nails of the fingers or the toes.

Pyococcic onyxis generally follows upon impetigo and especially upon panaris or whitlows; it extends beyond these conditions, its duration being much more prolonged, over a number of months. It begins as minute abscesses under the angle of the nail, which often dry without opening; they spread from one place to another in the vicinity in the nail-bed, more or less detaching the nail, which may itself become irregular, roughened and brittle, when the matrix has been invaded by the process. This onyxis is always accompanied by

perionyxis, namely redness, swelling and even pustulation of the peri-ungual folds.

The treatment consists of local baths with Alibour water and moist dressings; later on, in applications of yellow precipitate ointment or various tar preparations. When properly treated, this affection is readily curable.

Eczema of the nails is frequent, generally accompanied by eczema of the fingers and toes, as well as by perionyxis. The lesions are extremely polymorphous. There may be detachment of the nails with redness and subungual desquamation, longitudinal or transverse ridges with thickening, punctations, erosions, various deformities, etc. The treatment is that of eczema.

The *psoriatiform eczematides* and *psoriasis* give rise either to dotted cup-like erosions resembling the surface of a thimble, or to transverse and longitudinal striations, or to detachment beginning at one of the borders, etc.

A positive diagnosis of eczema or psoriasis of the nail cannot be made without taking into account the concomitant lesions, but it must be kept in mind that onychosis may precede the cutaneous manifestations and often survives them.

The treatment of psoriatic onychosis is very trying. Pyrogallol or chrysarobine salves, of 2.5 or even 10 per cent., may be employed. I prefer to paint the nails with a solution of one of these agents in ether or chloroform, or with some tarry solution, followed by covering with plaster or a varnish. Radiotherapy is sometimes very efficient.

In *pityriasis rubra pilaris* the nail is thickened, striated, literally like rush-pith, lustreless and yellowish; the thickening is due to a very hard although porous hyperkeratosis of the bed, which becomes conglomerated with the ungual plate.

In ordinary *alopecia areata*, but especially in the extensive and generalized forms, ungual lesions are frequent, consisting of dryness, white longitudinal striation, punctation (pitted nail), or of fissuring (onychorrhexis) crumbling and indentation.

In the severe primary *erythrodermas*, the nails become completely or incompletely detached (total or partial onycholysis) and in the last-named case, if a new nail is formed, it insinuates itself under the remains of the old nail. This partition of the nail is also seen after bruising, in syphilitic onyxis, etc.

In *pemphigus, chronic, foliaceous* and *hereditary*, and in the grave forms of Duhring's disease (Fig. 140), the nails are usually involved, although in very different ways. The nail may fall without becoming replaced; in this case, the peri-ungual folds become obliterated and the bed is perfectly smooth; or the nail is detached from before backward, or divided, or atrophied, or even onychogryphotic.

In *psorospermosis follicularis* [Darier's disease], the nails are longitudinally striated, furrowed and brittle.

Chronic *radiodermatitis* as an occupational disease gives rise to severe ungual lesions, consisting of fragmentation, destruction, exfoliation, or even total atrophy.

Onychosis of General Diseases.—All severe or even mild pyrexias, eruptive fevers, typhoid fever, pneumonia, anginas, epididymitis, etc.; severe traumatisms or operations; emotional shock, childbirth, etc., may give rise to a groove or a transverse or rather bow-shaped ridge on the nails. All the nails or only some of them, especially the thumbnails, are thus marked to a variable degree. [The severe sea-sickness of an ocean voyage is capable of producing this effect on the nails.]

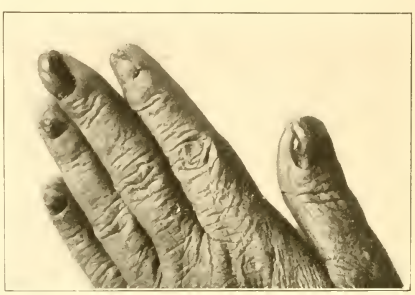


Fig. 140.—Lesions of the nails in a severe case of Duhring's disease. Definite loss on the index finger; atrophy on the ring finger and little finger; detachment, onychauxis and onychogryphosis on the thumb and middle finger.

This groove results from an arrest or momentary disturbance of growth; it emerges under the supra-ungual fold a few weeks after the cause which has produced it, advancing toward the extremity at the rate of growth of the nail, namely about 3 mm. monthly. It furnishes a valuable indication to recent disturbances in the health of the subject.

Chronic diseases, infectious or constitutional, may also interfere with the formation of the nails and cause *atrophy*, *koilonychia*, fissuration known as *onychoschizis*, simple hypertrophy known as *onychauxis* or *scleronychia* and *onychogryphosis*.

Diseases of the pleuropulmonary apparatus give rise to curvature with enlargement of the nails, known as the *Hippocratic nail*.

Syphilitic Onyxis.—Ungual lesions due to secondary syphilis are entitled to special mention on account of their diversity and importance. The condition is onyxis with or without peri-onyxis. Several varieties have been described by Fournier:

The nail may be cracked [*scabrités ungium*] and brittle at its free end; or detached from below upward (*onychoschizis*) with non-painful redness and desquamation of the bed and sometimes loss of the nail; or considerably hypertrophied (*pachyonyxis*), thickened, striated and blackish, without change of its general shape; or ulcerated (*helconyxis*); in the last-named case, the loss of substance, usually oval, crateriform and with lamellar borders, appears on the lunula and exposes the matrix or the bed of the nail, which is of a grayish-pink color.

The peri-onyxis is said to be scaly or horny when a squamous papule forms on one of the peri-ungual folds; inflammatory, when it consists of a dusky red, very persistent tumefaction; ulcerative, when a loss of substance with cut-out borders and a sanious floor develops, often as a semicircle, on the peri-ungual folds. The extremity of the finger is swollen and reddened; the nail usually falls out. Several fingers or toes are often attacked at the same time.

Fig. 141.—Syphilitic onyxis. At the thumb, pachyonyxis and onychoschizis; on the index finger, a large deep groove; on the other fingers, cracked, fissured, crumbling nails. The syphilitic infection of this patient was of ten months' standing.

These syphilitic onyxes and peri-onyxes (Fig. 141) are slightly painful, develop slowly and are subject to recurrence; they are apt to reappear in the course of the tertiary stage and prove very rebellious to treatment. Similar ungual lesions or more common ones are met with in congenital syphilis.

Dystrophic Onychoses and Onychoses of Nervous Origin.—Many nervous diseases involve the nails, notably the traumatic neuroses, syringomyelia, Morvan's disease, Raynaud's disease, tabes, hemiplegia, scleroderma, etc. Perhaps the general diseases referred to above act through the mediation of the nervous system.

The form of the lesions is extremely variable: simple falling-out, atrophy, detachment, fragility, deformity thickening, etc.

All kinds of onychoses, the nature and origin of which could not be discovered, have commonly been referred to a trophic cause. I yield to this tendency in grouping here the four following forms:

Leuconychia.—There exist two varieties of this affection: One is punctate and common in children, youthful individuals and women who take great care of their nails; it is characterized by small scattered or profuse white spots known under the name of *flores unguium*, *mendacia* (gift-spots), or sometimes by linear series of spots which appear toward the lunula and advance with the nail. Nervous disturbances, some intoxications, etc., have been held responsible; but in my opinion they are the result of slight traumatisms of the ungual matrix.

The other variety, *leukonychia totalis*, may be congenital or even hereditary; often it is acquired and occurs after a severe disease, or a neuritis, etc. There is also a striated variety, with alternately white and normal transverse bands.

The whiteness of the nail is due in both types to fine bubbles of air which have become infiltrated between the probably imperfectly keratinized ungual cells.

The patient should be instructed to avoid traumatism of the matrix of the nails, for example, in the manipulation of pushing back the supra-ungual cuticle [and the use of the pernicious cuticle knife]. The nails can be tinted with an alcoholic solution of eosin.

Onychogryphosis.—This name was given by Virchow to a deformity consisting of a sometimes enormous and very hard thickening, with a change in the direction of the nail, which becomes raised and curved on itself. The onychogryphotic nail of a low degree assumes the appearance of a very hard grayish or brownish claw, curved transversely and from before backward and raised from its bed by hyperkeratosis.

In a higher degree, the nail is entirely deformed, convex and twisted, resembling a ram's horn. It is implanted almost vertically on its bed, of a brownish color and marked at the same time by longitudinal striæ and transverse undulating striæ; the latter indicate the changed direction of the plane of the matrix. The nail grows slowly, but as its stony hardness prevents it from being cut, it often reaches from 3 to 4 cm. in length and exceptionally may attain a length of 10 and 12 cm. It is easily understood that it becomes troublesome in proportion to its size.

Onychogryphosis is observed especially at the feet, on the big toe and sometimes on the neighboring toes (Fig. 142). It is less common at the fingers, where I have, however, seen several cases of it. The pathogenesis proposed by Virchow, who holds pressure from the

shoes responsible for an irritation of the matrix, is therefore not tenable. Inflammatory lesions of the nail-bed, which were demonstrated by Unna, are perhaps secondary. It seems that a hyperkeratosis of the bed with hyperplasia of its papillary crests, according to Heller, raises and straightens the nail, which then grows thicker, but also more slowly. At any rate, this ungual dystrophy appears especially at a mature or advanced age; it is almost regularly present on varicose limbs, bearing ulcers or the seat of elephantiasis; or it may coincide with chronic rheumatism, arteriosclerosis, neuritis, leprosy, etc.

The palliative *treatment* consists of scraping, sawing, or tearing off the nail; it may first be softened by means of potash or salicylic acid. In order to prevent re-growths, it is necessary to excise the ungual matrix.

FIG. 142.—Onychogryphosis of the first three toes.

Onychorrhexis.—Dubreuilh and others give this name to longitudinal striation with fragility of the nails.

In pronounced cases, the nail is lustreless, cracked, roughened, thinned and broken. A few or all the nails may be involved. Onychorrhexis may date from childhood, or it may develop as a sequel of nervous disturbances, lichen planus, alopecia areata, etc. The nails may be painted with collodion to remedy the painful cracks and fissures.

Onychoschizis.—This term ($\sigma\chi\iota\zeta\epsilon\iota\nu$ = to separate) designates the detachment of nails from their bed. This separation is, as a rule, progressive and more or less rapid, with periods of remission and exacerbations. It begins under the free border and gradually ascends as far as the lunula; a single finger may be involved, or usually several fingers and toes are attacked simultaneously or progressively. Sometimes the separation is evidently dependent upon a subungual hyperkeratosis with accumulation of solid or friable horny material.

Onychoschizis, like onychorrhexis and a considerable number of other changes of the nails, mentioned above, are undoubtedly not always of the same etiology and significance. Every case should be traced to its cause, if possible.

General Diagnosis of the Onychoses.—Sometimes the objective features of ungual lesions, or the circumstances and coincidences are such that they can be referred to a definite cause from the start. Not infrequently, however, in clinical cases the etiology is neither apparent nor easily discovered. A hasty diagnosis of "trophic disturbance of the nails"—as is only too frequently made and merely amounts to an admission of ignorance—should be refrained from in such cases. When confronted with an onychosis of obscure character, the physician must pass in review the different classes of lesions enumerated in this chapter, in order to find out where it belongs.

It is well to remember the following facts: Malformations of the nails of congenital origin, or those connected with familial and hereditary dystrophies may appear a fairly long time after birth; permanent deformities of the nails may result from traumatisms which have damaged their matrix; onychomycoses of indefinite duration may occur without tinea, sycosis or epidermomycosis, and require microscopic examination; the onychoses of the dermatoses, pyococcia, eczema, eczematides, psoriasis, lichen planus, Duhring's disease, etc., not infrequently persist after the cutaneous manifestations have disappeared and may exceptionally precede them; syphilis and congenital syphilis require very careful investigations and sero-diagnosis.

Finally, the discovery of chronic constitutional diseases (diabetes, etc.) or infectious diseases (tuberculosis, etc.), as well as nervous diseases, even abortive or incipient, may sometimes supply diagnostic indications and suggest the proper treatment.

A systematic review of this kind will considerably reduce the number of cases described as *trophic disturbance of the nails*, or *trophoneurotic onychosis*.

The *treatment* must frequently utilize either keratolytic or reducing agents. Often, even in cases of enigmatic character, considerable benefit is obtained from high frequency currents and especially from radiotherapy.

CHAPTER XXII.

HIDROSES.

The name hidroses (ἵδρωϛ = sweat) may be applied to functional disturbances of the sweat-secretion and to the primary organic lesions of the sudoriparous apparatus.

The *sweat glands* are epithelial tubes whose deep or secretory portion is rolled up as a *glomerulus*, situated in the lower layers of the corium or the upper layers of the hypoderm; their ascending excretory portion, the so-called *sweat-duct*, passes directly through the derma and twists as a spiral in the epidermis. This channel opens obliquely at the surface through a *pore*. This arrangement of the pore and the sweat-channel and, moreover, the direction of the fluid which passes through them, oppose the penetration of dust and microbes into the interior, so that infections of the skin rarely occur by this route.

The sweat-glands are distributed in unequal numbers over the entire integument, including the palmar and plantar regions, where they are indeed especially abundant. In the axillary fossæ and in the anogenital region, they are of a special type, their secretory portion here having a very large lumen and serving as a receptacle for the sweat.

The *sweat* is a watery fluid, of an odor varying according to body regions, individuals and races; usually of an acid reaction except in case of profuse sweating; in the axillæ and in the groins, the reaction is normally alkaline.

The quantity of sweat excreted in a given time varies enormously, and is influenced by temperature, exercise or rest, amount of beverages, action of the nervous system, as well as some pathological conditions and medicinal agents. The average quantity is estimated at about a litre in twenty-four hours.

Under normal conditions, the sweat evaporates in proportion to its production. Neither this *insensible perspiration* nor profuse sweating act as emunctories, but they greatly contribute to the regulation of the body temperature. Another function of the sweat is the maintenance of a moist and supple horny layer, as well through its watery content as through the emulsified fat contained in it.

The chapter on hidroses is not of particular interest to the dermatologist. A distinction must be made between functional and organic hidroses:

1. Disturbances of the sweat secretion, or *functional hidroses*, belong to a considerable extent to the domain of general medicine.

2. Organic lesions of the sweat-glands, or *organic hidroses*, are on the whole rare.

Among the latter, *hidrocystoma* and *hidradenoma* will be described with the tumors; among the keratodermas, in a preceding chapter, mention has been made of *porokeratoses* or punctuate keratoses, which are without sufficient reason interpreted as ostio-sudoriferous keratoses. *Dysidrosis* and *miliaria* likewise have no definite relations with the sweat structures. The following discussion will accordingly be limited to sudamina, hidradenitis and miliary abscesses.

FUNCTIONAL HIDROSES.

The anomaly concerns either the quantity, or the odor, or the color of the sweat.

Anidrosis.—Very exceptional cases have been reported of individuals who do not perspire and therefore imperfectly regulate their temperature, as a result presenting symptoms of heat-stroke on even moderate exposure to the sun in summer-time. A striking observation of this kind was recently published by R. Lutenbacher (May, 1917).

The sweat is very scanty in persons suffering from diabetes, myxedema, cachexia, ichthyosis, as well as in senile degeneration of the skin. Sweating is temporarily absent in several chronic erythrodermas, on the patches of psoriasis and in a considerable number of other eruptions.

Hyperidrosis.—Individual differences in the amount of sweat excreted are such that hyperidrosis is never more than relative.

The profuse transpirations which occur in the course or at the decline of the pyrexias, sweating or miliary fever, acute rheumatism, the sudoral form of typhoid fever, pneumonia, grippe, septicemias, etc., the sweats of phthisis, gout, or the moribund do not belong to the realm of this discussion.

In a considerable number of organic or functional nervous diseases, in hemiplegia, tabes, neuritides, wounds of the nerves, lesions of the sympathetic, neurasthenia, exophthalmic goitre, etc., there occur profuse sweats, generalized or partial, permanent or in attacks. This is suggestive of a nervous pathogenesis in the so-called essential hyperidroses. Moreover, these are preferably observed in highly emotional individuals, or associated with obesity or auto-intoxication. The sweat may exude all the time or, on the slightest effort or trifling emotion, moisten the surface of the skin, sometimes flowing in profuse drops. Maceration of the epidermis in the folds is a common sequel.

Hyperidrosis may be general, or more frequently partial, when it is known as *ephidrosis*. There is, for instance, a total facial ephidrosis, or hemifacial or localized ephidrosis. The secretion in these patients may occur as a reflex under the influence of mastication or certain gustatory sensations, among others that of vinegar or chocolate.

It is a familiar fact that bald persons usually transpire freely from the scalp and face; I believe that this hyperidrosis is connected with kerosis and dependent upon its multiple causes (p. 196).

The sweat of kerotic individuals is sometimes charged with such an abundance of emulsified fluid fat as to cause grease-spots on pillows, hats, paper placed on the skin, etc. These cases are designated by the name of *hyperidrosis oleosa*. The condition is probably a combination of hyperidrosis with seborrhea oleosa (p. 393).

The peculiar affection which bears the name of *granulosis rubra nasi* seems to be regularly associated with hyperidrosis and is probably a result of the latter. It is observed especially in children and is characterized by small pinkish or red miliary papules on a lavender and cold background; the lesions occupy the nose and sometimes the lips or the chin; they become less marked with advancing years.

The extremities are constantly moist or even wet with sweat in some individuals. When the hands are affected, a number of occupations are rendered impossible; at the feet, the excessive sweat macerates the epidermis, makes the skin very vulnerable, markedly impedes walking and often becomes offensive. Hyperidrotic extremities are rarely warm and congested; as a rule, they are cold, acro-asphyxiated and flabby, or of a waxy white color.

Hyperidrosis of the articular folds, especially of the axillæ, is extremely common; many obese, gouty, rheumatic or nervous individuals suffer from this condition. Axillary hyperidrosis may lead to lepothrix and to chromidrosis. It predisposes to intertrigo, to deep abscesses, etc.

All dermatologists are familiar with *hyperidrosis nudorum*, a free flow of sweat from the axillæ seen in patients who are stripped and exposed before an audience. In these cases, an excretory rather than a secretory reflex is concerned; under the influence of cold and emotion, the smooth muscle fibers of the large axillary glands become contracted and empty their contents.

Bromidrosis.—This name is applied to the offensive sweats which occur especially at the feet, sometimes in the axillæ or groins, constituting a most distressing infirmity.

Usually, but not invariably, bromidrosis is connected with hyperidrosis and is a complication of the latter. It is possible for fatty acids or ammoniacal compounds to become eliminated in excess through the sweat, but as a rule the offensive odor is derived from secondary decomposition.

In the *treatment* of hyperidrosis and bromidrosis, it is important not to neglect the correction of whatever may be deficient in the general hygiene of the patients. Locally warm or lukewarm baths are recommended; alcoholic rubs, for example with camphorated alcohol, or with alcohol containing iodin or tannin; and applications of ichthyol or various powders.

Cases of *offensive sweating of the feet* require scrupulous cleanliness, a frequent change of foot-wear, the wearing of shoes permeable to evaporation. Baths, alcoholic rubs, bland powders charged with salicylic or tartaric acids, or with bismuth subnitrate, are often inefficient. Formol, in all its forms, is dangerous.

A good treatment of plantar hyperidrosis and bromidrosis consists in painting the parts daily with a solution of potassium permanganate (1 to 10 per 1000); or with iron perchloride solution with 25 per cent. of glycerin.

Still better results are obtained by painting the parts, daily at first, then at longer intervals, with a solution of chromic acid (2 per cent., or even 5 or 10 per cent.) followed by dusting with talcum-powder; but these applications must be very cautiously made.

Against palmar hyperidrosis, radiotherapy has been shown by Sabouraud to be the cardinal remedy; sometimes, a single application of 5 units H. is sufficient. The same treatment is appropriate for *granulosis rubra nasi*.

Chromidroses. — The problem of the chromidroses or *colored sweats*, to which several publications were devoted by Le Roy de Méricourt, still remains very obscure. Many of the reported cases are doubtful or simulated, but a few authentic observations are on record.

The colored sweats are always regional, partial, or even very circumscribed; they may be blue, red, or black, or sometimes yellow; green, etc. The color as a rule is the result of oxidation on contact with the air of a chromatogenic agent contained in the sweat, which then becomes deposited on the epidermis in a pulverized form.

A blue coloration is apparently due either to pyocyanin and coexistent with blue suppuration, or to indican. A red color is more common, especially in the axillæ; it seems to be of microbic origin and is frequently associated with lepothrix. Dry erythidroses with adherent pigment have also been reported. The case of melanidrosis which was studied by R. Blanchard on the eye-lids of a thirteen-year-old boy confirms beyond a doubt the existence of this form. The black pigment which he saw becoming deposited around the sweat-pores was examined by Maillard and found to be related to the choroid pigment.

Hematidrosis.—It is conceded that an oozing of blood derived from the capillaries may take place on the intact skin by way of the

sweat pores, hence the name which has been given to this phenomenon. The majority of the cases reported by the ancients, of bloody sweats under the influence of extreme emotional distress, are undoubtedly mythical. Perhaps the condition actually occurs in the course of severe infections, or in neuropathic individuals, in the presence of purpura, or as a vicarious menstruation. I am not acquainted with any convincing observations.

ORGANIC HIDROSES.

In a large number of dermatoses, the sweat-glands are affected together with the neighboring tissues, or secondarily. Primary and idiopathic affections of the glomeruli and sweat-ducts are on the contrary rare.

It has previously been stated that in dysidrosis the sweat-glands play no part; also, that *miliaria rubra* and *alba*, although designated as sudoral eruptions on account of the conditions under which they are usually observed, are in reality a miliary impetigo without constant relation to the sweat glands (pp. 85 and 87).

Sudamina [**Miliaria Crystallina**] is the name applied to an affection characterized by extremely minute bullous elevations of the horny layer, containing a clear watery fluid and without a congested base.

The sudamina are the average size of a farina grain. Sometimes they are scattered in very variable numbers, sometimes grouped or even confluent in small blebs; they are observed on the trunk, notably on the flanks and abdomen, as well as on the flexor surface of the limbs. They appear suddenly without pruritus, altogether or in successive crops. Their duration is ephemeral; they dry out, the epidermis peels a little and all is well again. It is not impossible for a few small vesicles to acquire turbid contents and a slightly reddened base, as the sequel of a secondary infection which transforms them into miliary impetigo; but this is an exceptional occurrence.

Sudamina have been encountered in the course of severe pyrexias, such as acute rheumatism, typhoid fever, scarlatina, at the crisis of pneumonia, or as a phenomenon preceding death. They are also observed to occur under occlusive dressings in the hot season, or in the course of treatment with keratoplastic agents.

A sudamen, as shown by J. Renaut, is a small bleb situated in the horny layer or immediately below it, on the course of a sudoriparous canal. It may be interpreted as the mechanical result of splitting of this layer under the influence of a gush of sweat occurring after a temporary anidrosis with stagnation of the horny layer, or exaggerated keratinization. The sudaminal contents are therefore probably

sweat, which by some authors was found to be alkaline or neutral, by others, including Jadassohn, invariably acid.

No importance, not even a prognostic value, can be attached to this trifling lesion.

Hidrosadenitis.—Verneuil (1864) described as *phlegmonous hidrosadenitis* or *sudoriparous abscesses* an affection commonly observed in the form of tuberous abscesses of the axilla (Velpeau) and which according to him may also be localized at the circumference of the anus, at the external auditory meatus, or even become disseminated.

This condition is a form of staphylococcic pyodermatitis which will be described elsewhere in this book. It is usually admitted to result from an infection by way of the sweat-channels, although this fact cannot easily be demonstrated directly. [This disease should not be confused with hidradenitis suppurativa (Pollitzer), which is now included in the papulo-necrotic tuberculides.]

Miliary Abscess of Little Children.—This condition has been known for a very long time and was already distinguished by Alibert from furunculosis. It occurs in neglected or greatly weakened infants suffering from impetigo, eczema of the buttocks, gastro-enteritis, bronchopneumonia, etc., and consists of multiple scattered resistant intradermic nodules, all of about the size of a small pea. On puncture a creamy pus is evacuated, their cavity is more or less deeply situated in the cutis. They occur in crops, sometimes repeated; the general phenomena are very variable.

The pyemic character of these abscesses was suspected in the past. However, Escherich and Longard surmised and Lewandowski (1906) demonstrated that they are in most cases the result of a staphylococcus aureus infection; the infection takes place through the sweat-pores, a point of lessened resistance in the infantile skin. The almost invariable coexistence of small intra-epidermic pustules the size of a pin-head has been demonstrated, analogous to the ostiofolliculitis of Bockhardt, but due to infection of the sweat-pores and designated by Lewandowski as *periporitis*.

As to *treatment*, incision of the abscesses is recommended, next sweating followed by sublimate baths of 1 per 10,000. Zinc sulphate baths of the same strength, or washes with Alibour water, would undoubtedly prove equally efficient.

PART II.

NOSOLOGY OF THE DERMATOSES.

In the first part of this book I have discussed the morphology of the dermatoses with a comparison of those related either by a common eruptive lesion or pathological condition of the skin, or by a conformity of localization in the same cutaneous or mucous territory or in the same organic adnexa of the epidermis.

In the following part the dermatoses will be grouped according to their etiology, considering together those which are dependent upon the same kind of causes.

CHAPTER XXIII.

ARTIFICIAL DERMATITIDES.

Inflammations of the skin resulting from the injurious action of a mechanical, physical, or chemical cause are called *artificial dermatitides*.

Their clinical features are extremely variable; they may assume practically all the forms of dermatological lesions, especially those of erythema, urticaria, purpura, eczema, blebs, gangrene, etc.

The severity of the lesions, their superficial or deep character, their more or less rapid or slow onset, their ephemeral or prolonged duration, depend in part upon the cause and in part upon the soil on which it has acted. The *cause* may in itself be more or less markedly injurious; its action may have been more or less intense or persistent. The soil, namely the damaged skin, in its turn presents variations in vulnerability and capacity of reaction according to the patient and the region of the body.

Those relatively rare cases must be separately classified in which there exists what Brocq calls (July, 1915) a diminished resistance of the skin, which may be congenital or regional; this abnormal vul-

nerability—which is the basis of congenital pemphigus, hydroa vacciniforme and xeroderma pigmentosum, but which I have observed also outside of these affections—often manifests itself only toward a certain class of injurious agents (traumatism, light, etc.).

In normal individuals, it may be stated that the influence of the patient's individuality, his personal equation, what is known as the morbid predisposition, is relatively slight in the case of most dermatitides of mechanical and physical origin; whereas this predisposition plays on the contrary a leading part in the pathogenesis of dermatitides of chemical origin, or toxidermas. It will be more fully considered in the section devoted to the latter.

It must be understood that there is no absolute and constant agreement between cause and effect in the artificial dermatitides. From this rule are derived the two following corollaries:

The same injurious agent may give rise, in different cases, to fundamentally dissimilar eruptions. The dose, the avenue of invasion, still other factors and especially the individual predisposition strongly modify the consequences of the same cause. In this way, to quote an immediate example, the absorption of an iodide may give rise, in different cases, to erythema, urticaria, purpura, bullæ, pustules, etc.

Inversely, dermatitis of identical appearance may be derived from entirely different injurious causes. A bullous dermatitis, for instance, may be the result of a burn, the application of a blistering or caustic substance, or the absorption of a medicinal agent such as an iodide or antipyrin. It could not be otherwise, for while injurious agents are extremely numerous, the modes of reaction of the skin are limited in number.

In dealing with a given cutaneous lesion, the clinician is therefore often puzzled at its origin. It is precisely this difficulty which lends interest to the artificial dermatitides which are extremely common.

I shall successively review (1) dermatitides due to mechanical causes; (2) dermatitides due to physical causes; (3) dermatitides due (or apparently due) to chemical causes, which may be called *toxidermas;* these should be subdivided into two groups, according to their external or internal origin. It seems advisable to add to the description of the latter a comment on the autotoxic dermatoses, which although not artificial may pass as toxidermas.

DERMATITIDES DUE TO MECHANICAL CAUSES.

Traumatisms of all kinds, when slight, may give rise to erythema (Chapter I). More severe traumatisms cause contusions, wounds, etc., in short, surgical complications.

The following belong to the domain of dermatology: Traumatic

eczema (Chapter IV); excoriations from scratching and the epidermic hypertrophy known as *lichenization;* the latter will be discussed in the chapter on Pruritus (Chapter XXIV); certain traumatic alopecias; and furthermore, the effects of repeated or prolonged pressure.

Strong and persistent pressure on some part of the skin, like that exerted by a badly fitting plaster apparatus, gives rise at first to erythema, then to phlyctinization, finally to ulceration and even to gangrene.

Repeated pressure with friction on a thick epidermis, like that produced on the foot by hard shoes during a long march, or on the hand by the manipulation of some tool or instrument, causes the appearance of bullous elevations known as traumatic blisters, well known to soldiers, mountain-climbers, boatmen, acrobats, etc.

Attention is here called to the special vulnerability of the skin which constitutes *congenital pemphigus* (p. 191).

Callus.—Callus or *callositas* represents a chronic traumatic dermatitis with thickening of the derma and hyperkeratosis. Callosities are observed on the hands of laborers, where their seat often betrays the occupation of the individual (diggers, gardeners, blacksmiths, tailors, etc.); on the feet, notably on the instep and at the ankles; moreover, on the prominence of the head of the first metatarsal in case of hallux valgus, where they contribute toward the formation of bunions; on the ischial bones of cavalrymen, etc. In short, callosities may form at any point on the body.

Callus is a yellowish or pigmented prominence of variable extent, round or oval, with gently sloping borders, thick and solid on palpation. Being a defensive reaction of the integument, the callus is not painful either spontaneously or on pressure unless it is inflamed. In this case—the "durillon forcé" of Chassaignac—a blister may form underneath the thickened epidermis; when it becomes infected, the subepidermic abscess often gives rise to lymphangitis with fever, so that the patient is obliged to stay in bed.

The only *treatment* of simple callus consists in the suppression of its cause. Moist dressings and rest are the measures most to be recommended in case of inflammation.

Clavus.—A corn or clavus is a local traumatic hyperkeratosis with inflammation, then atrophy of the cutis, occurring only on the feet, at points exposed to chronic pressure from badly fitting shoes. It is situated especially on the prominence of the phalangeal articulations of the toes, particularly on the little toe, or on the metatarsal prominences and sometimes on the sole of the foot. Its dimensions vary from those of a lentil to a small bean. Corns are always painful, either spontaneously in damp weather, or on pressure when they are not trimmed.

Corns differ from callosities in the much greater thickness of their horny layer which projects as a smooth or scaly prominence and is deeply embedded in the cutis; the latter is depressed like a funnel and inflamed or atrophied. In the center of the horny mass a softer, white, medullary portion can often be distinguished, formed by poorly keratinized cells. Underneath the corn the malpighian layer is thinned and the papillæ are obliterated. Occasionally the center of the corn presents a papilla with dilated vessels projecting into the horny mass. The so-called "root" of the corn has no actual existence and its extraction by quack chiropodists is nothing but a trick.

Interdigital corns, known in France as "œil de perdrix" (partridge eyes), are softened as the result of maceration [soft corns].

Corns must not be confused with plantar warts or with mal perforans.

To be afflicted with corns may mean an extremely distressing if minor infirmity, for which patients are apt to neglect seeking professional advice. Left untreated and irritated by pressure, corns may become the seat of an inflammation, a blister, an abscess, etc. When badly cut, they often give rise to lymphangitis.

The popular *treatment* consists in trimming the hyperkeratotic layer with a razor at regular intervals before it again becomes exuberant and painful, as well as in avoiding all local pressure. The horny mass may also be softened by nocturnal moist dressings, or by keratolytic agents such as salicylic acid plasters, or salicylated collodion, which permits the removal of the corn in a warm bath. Without patient and persistent treatment, a relapse is very probable.

DERMATITIDES DUE TO PHYSICAL CAUSES.

Heat, cold, solar and electric radiations, *x*-rays, beyond a certain limit, are injurious to the human integument. They give rise to a series of lesions, that range from erythema to gangrene. Superadded pyococcic infections frequently complicate the clinical picture.

Aside from these general data, which apply to all, it is noteworthy that especially in regard to light radiations, certain individuals are much more susceptible than others (solar erythema, pigmentations); also, that there actually exist special pathological conditions (herpes vacciniformis, xeroderma pigmentosum) characterized by disproportionate reactions to the effect of light.

Burns.—Burns may be produced by burning or incandescent bodies or by heated substances, solid, liquid, or gaseous. Lesions caused by bright light, electricity, or caustic agents, are often placed in the same group.

Three degrees of burns are recognized:

In the *first degree*, the lesions consist of *erythema*, with severe pain, heat, swelling, representing erythema *a calore*. In a few days, after desquamation, the skin is well again.

The *second degree* is characterized by *bullæ*. These form either immediately, the vaporization of the intramalpighian plasma having raised the horny layer under which a certain amount of serum collects; or secondarily, after a few hours, through the mechanism of superficial or deep bleb-formation. The contents of the bullæ, which vary greatly in size, are lemon-yellow, fluid or gelatinous. The pain is extremely severe. The bullæ having ruptured, the fluid escapes and a bright, red, shining surface appears under the epidermic membrane; or they may shrivel and dry in crusts; or they may become infected and suppurate. In the last-named case, a cicatrix may persist. The course of healing lasts one or two weeks.

In burns of the *third degree*, there is sloughing, coagulation and necrosis of the cutis and sometimes of the subjacent parts. Blisters appear only on the borders of the slough which in different cases is yellowish or brown, or dry and anesthetic. The pain may be less severe than in a burn of the second degree, on account of destruction of the nerve-ends. The course, of very variable duration according to the depth and extent of the burn, the region affected and the evactual complications, is that of a non-progressive gangrene. The cicatrix is often vicious.

Very extensive burns, like those of children who have tumbled into a boiler, or of soldiers struck by an incendiary shell, or those which I saw after the fire of the Puteaux powder mills, where the unfortunate women workers were burned from head to foot except on the parts protected by the corset, are accompanied by grave general symptoms. Chills, fever or rather a lowering of the central temperature, collapse or delirium, bloody vomiting, melena, albuminuria and pulmonary complications are due in such cases for the most part to toxic products resulting from the alteration of the blood in the cutaneous vessels, under the influence of heat; in part to the nervous shock and finally to superadded infections. Burns involving over one-third of the total tegumentary surface are usually fatal.

The *treatment* of burns consists in cleansing the skin, in the evacuation of the blisters without pulling off the epidermis, in the application of sterile occlusive dressings. Packing in aseptic cotton, after painting with dilute tincture of iodin (1 to 2) constitutes an excellent procedure of immediate treatment; if necessary, the clothing must first be cut away.

Among the countless topical applications which have been recommended, including even raw scraped potato and gooseberry

jelly, which actually relieves the pain, the preference must be accorded to oil- and lime-water liniment, vasolanolin, naftalan, or better still, to the special oils named pyrolcol and phlyctol, which are highly analgesic. The burn is covered with sterilized compresses soaked in one of these products and with a thick layer of cotton. The dressings should be changed as rarely as possible, except in case of suppuration. Ichthyol, glycerinated thiol, the salve of J. Lucas-Championnière and the product named ambrine, can also be recommended. Picric acid which has found so much favor, should be rejected, for it is sometimes dangerous.

In case of very extensive burns, permanent lukewarm baths give the most relief. It goes without saying that an injection of morphin should not be refused to these unfortunates in their atrocious sufferings. Camphorated oil and adrenalin by subcutaneous injections and enemas with physiological salt solution, are indicated in grave cases.

Frost-bite.—Lesions caused by freezing differ in many respects from those due to extreme heat. Cold requires a longer time for its action. Immediate pain is practically absent and nothing is felt but an unpleasant numbness; pruritus and an intolerable burning sensation being delayed until the reëstablishment of the circulation. It is almost invariably the extremities that are attacked by frost-bite; the feet, the hands, the ears and the nose. In the first degree the region is of a cadaveric pallor, followed by intense congestion. The second and third degree, bleb-formation and sloughing, usually exist together, the lesions attaining their maximum at the periphery, at the tips of the toes, the fingers, etc. The integument is at first of a waxy color, the region is anesthetic and lifeless. It is impossible to determine to what depth the tissues are affected till after a few days.

The frozen parts must be very cautiously and progressively warmed, preferably by rubbing them with snow; they may be covered only very gradually and the surrounding temperature raised very slowly. The subsequent course and the treatment are the same as in gangrene of any origin. The physician's attitude should be as conservative as possible and surgical intervention be restricted to the removal of a sequestrum. Frost-bites may lead to grave mutilations.

Chilblains and *trench-foot* have been discussed elsewhere in this book (pp. 34, 35).

Radiodermatitides.—The *x*-rays as well as radium, mesothorium, etc., although causing no sensation whatever at the moment of application, may give rise to a series of cutaneous manifestations, the extreme degrees of which are very grave. However, as there seems to be no individual idiosyncrasy toward these radiations,

these disturbances can always be avoided. They depend exclusively upon the dosage and quality of the rays. It is therefore of the utmost importance for radiologists to employ all measuring instruments belonging to an outfit of this kind, to be entirely familiar with the peculiarities and strength of their apparatus, to calculate accurately without mistakes the distance from the anticathode to the skin as well as the time of the exposure.

Unfavorable sequelæ of *x*-ray treatment and the occupational radiodermatitides of radiologists themselves will be discussed separately in the following.

Therapeutic Radiodermatitides.—The cutaneous reactions due to the *x*-rays do not appear until after a period of latency or incubation, lasting from eight to twenty days, usually from fifteen to seventeen days. The slight redness which may supervene on the same day or the day after the session is an erythema presumably due to the light rays and is of no importance.

Alopecia of the scalp regularly appears upon a dose of 5 H, so that Sabouraud was enabled to formulate the radiotherapeutic treatment of the tineas. On the face and elsewhere, alopecia is not a constant result and is often complicated by pigmentation and cutaneous atrophy.

Beyond a dose of 5 H in a single session, or if exposures of the same point are repeated beyond the ratio of tolerance as established by experience, there is danger of a series of manifestations, to be enumerated in the following: It must be kept in mind in this connection that the *x*-rays have a cumulative effect; that nevertheless there is a loss with time, differently estimated by authors, but which can be rated at 1½ H weekly; finally, that certain regions are especially susceptible, notably the back of the hands and feet, the bridge of the nose and the forehead—perhaps all points where the integument rests upon a bony surface.

The *erythema*, the first degree of radiodermatitis, punctiform and pink at the onset, later on becomes a uniform dusky or purplish red; it gives rise to severe itching, then to desquamation, lasting for a few days to a week, rarely longer.

The erythema is usually followed by *pigmentation* which may last for months, varying greatly in different individuals.

Blebs, scattered, grouped, or confluent upon an erythematous base, may develop after a more energetic exposure and are often the forerunners of ulcers or sloughs.

Ulceration usually burrows on the floor of a bleb which has suppurated. It is dusky red, smooth or slightly granular, with gently sloping borders, of extremely variable shape and extent, with scanty serous oozing. It is accompanied by burning, tearing, lancinating pains, often of extreme severity, preventing sleep and

constituting a serious torment. Radiodermatitic ulcers take a sluggish course, first progressive, then slowly retrogressive; they often last several months or even years. I have pointed out that sometimes they may simulate superficial epitheliomas, biopsy being necessary for a diagnosis.

Sloughing develops primarily, or under a blister, or around an ulceration. The slough is white, then brown or black, hardly encroaching upon the cutis or on the contrary also involving the cellular tissue, the tendons, the aponeuroses, etc., briefly, all the subjacent parts. It causes frightful pains, radiating to a distance, of a neuritic character. The course is very slow; demarcation and elimination are long delayed. Relapses, with reproduction of sloughs at the periphery or below are not uncommon. A cure requires months and may be followed by recurrences *in situ*. A very instructive case of ulcerative radiodermatitis came under my personal observation and was published in the *Annales de Dermatologie*, October, 1913. An ulcer on the breast of a young woman, occurring six months after a final *x*-ray session, healed in five months; it reappeared spontaneously, without a new radiation, over eleven years afterward and at this time still presented the histological structure of a recent radiodermatitis. It must be concluded that the pathological process persists practically indefinitely.

The patches of *sclerous radiodermatitis* which follow upon an ulcer, but may also develop as the sequel of a simple erythema or bleb, have a pathognomonic appearance. There is an indistinctly outlined spot of white, tense and indurated sclerosis, smooth and interspersed with serpentine or arboreal telangiectases; or there may be an undulating, scaly, *lardaceous* surface, mottled with pigmentary spots and purplish macules, adherent and not readily folded; finally, a thickened and deformed genuine *cicatrix* may be present. These three degrees of lesions may coexist in concentric zones.

Observers have been impressed with the analogy between the patches of white or lardaceous sclerosis in radiodermatitides and scleroderma in patches, which, however, is more sharply outlined; and also with xeroderma pigmentosum and senile degeneration, but the last-named affections are diffuse and regional (Chapter XVII).

Complications due to overexposure to the *x*-rays should no longer occur at the present day. Before any new application, it must be kept in mind that the patient may forget to mention, or try to conceal a recent *x*-ray treatment. Careful inquiries must therefore be made.

Occupational Radiodermatitides.—Radiologists, unless carefully observant of the most scrupulous precautions, are exposed to a diffuse, insidious, chronic and progressive radiodermatitis, which is

always grave and may become fatal. The hands, sometimes the face, are the parts naturally most affected. The fingers at first become cyanotic; the epidermis becomes dry and peels off, the skin becomes swollen and cracked, retracting especially around the nails which likewise become furrowed, split, break and exfoliate. The patient has a constant feeling of constriction.

The appearance, which is at first suggestive of frostbite or of dysidrosis, later on approximates that of the eczematized prurigos; but the mixture of atrophy, telangiectasis, verrucosities, etc., on the other hand, lends the clinical picture an analogy with xeroderma pigmentosum and with arsenical keratosis.

The occurrence of *epithelioma* of the papillary, later the lobulated type, is not very rare in these chronic radiodermatitides. It cannot be stated outright that *x*-rays give rise to cancer, but it must be admitted that their cumulative effect leads to a cutaneous dystrophy on which epithelioma more easily develops in the same sense as on the senile, arsenical, xerodermic dystrophies, or on cicatrices in general. The pioneer radiologists who worked at a time when the remote dangers of the *x*-rays were still unknown, have nearly all expiated their unwitting lack of caution with severe mutilations and in some instances with their lives.

Treatment.—This must be primarily prophylactic. The application of *x*-rays and radium requires scrupulous attention and considerable experience and caution. It must moreover be kept in mind that those cutaneous regions which have once been overexposed retain indefinitely a remarkable vulnerability toward mechanical, physical, chemical and medicinal noxious agents and especially a susceptibility to *x*-rays; they suggest fire smouldering under the ashes.

Erythema or blebs are treated like burns. Against the pigmentations, hydrogen peroxide or naphthol creams may be employed. Occupational radiodermatitides, sclerotic radiodermatitides and ulcers do well under repeated baths and moist occlusive dressings, notably a chamomile infusion mixed with potash, 1 per 10,000; inunctions with vaselin-lanolin cream and lead-water, or with glycerol of starch. Stimulating, antiseptic, analgesic topical applications and reducing agents in general should be avoided. Oily dressings are often readily tolerated.

Static baths, high-frequency currents and even heliotherapy have been recommended, but cannot be unconditionally endorsed. In certain cases, the excision of an ulcer may be indicated when this is feasible, or the destruction through superheated air. [Radical surgical excisions of deep *x*-ray ulcers followed by grafting not only gives prompt relief to the intolerable pains but is the best insurance against the subsequent development of epithelioma.]

Of recent years, especially in America, attempts have been made to treat the spots of keratosis and the incipient epitheliomas of radiologists, through applications of radium. This paradoxical therapy, which has the advantage of being painless, seems to have yielded really successful results.

TOXIDERMAS.

The number of substances of all kinds which may damage the skin is so great, their effects are so multiple and variegated that an entire volume might be devoted to this subject without exhausting it. I shall limit myself to a consideration of those features which have the most important practical bearing.

Etiology.—In the etiology of the toxidermas we have to deal not only with poisons, a group of substances actually incapable of definition, but also with countless medicinal agents, the list of which is extended every day through the achievements of chemistry, as well as many products employed in the industries, in the household, for the care of the person, etc., and finally a certain number of foods and drinks.

According as the injurious substance has acted through application or friction on the skin, or by its having been ingested and absorbed, a distinction is made between: *External toxidermas*, or the *direct artificial eruptions* of Bazin, on the one hand; and the *internal toxidermas*, or *indirect artificial* or *pathogenetic* eruptions of Bazin on the other.

In the latter case, the digestive mucosa most frequently serves as the avenue of entrance; exceptionally, the urogenital, respiratory, or even the conjunctival mucosa may be responsible; finally, the injurious substance may have been introduced by the hypodermic route, which is becoming more and more used for therapeutic purposes; eruptions due to sera form an interesting group of this class.

Symptoms and Diagnosis.—A few general remarks will suffice in this connection.

Three points have to be settled: (1) The presence of a toxiderma; (2) its external or internal origin; (3) the discovery of the injurious substances.

1. As there exists no characteristic sign of *toxiderma in general*, the prudent physician will make it his rule, when dealing with any dermatoses the diagnosis of which is not at once evident, to think of the possibility of an artificial eruption and to pursue his inquiry in this direction.

It would be a mistake to assume that the eruption is the entire disease in the toxidermas. Accessory pathological phenomena are frequent. Aside from the painful, burning, tense and espe-

cially itching sensations which usually accompany the eruptions, general disturbances may also be observed, such as malaise, excitement, headache, insomnia, various digestive disturbances, a pale or yellowish complexion, etc. Fever may be associated with these symptoms; it is usually moderate and ephemeral; exceptionally it may be very high.

The internal toxidermas are accompanied in certain cases by *enanthema* of the buccal, pharyngeal, nasal, conjunctival, genital mucosæ, sometimes even by edema of the larynx; this may be notably the case in iodism, hydrargyria, etc. It follows from this fact that the differential diagnosis of certain toxidermas from the eruptive fevers or from various infections derived from a mucous membrane, may present very serious difficulties.

2. The *external* or *internal* origin of a toxiderma is sometimes evident at the first glance; the type of the eruption, its topographical distribution, its localization or generalization may furnish valuable indications in this respect. In other cases, however, this question cannot be settled off hand. It may happen that a substance applied to the skin acts both locally and by absorption. Hence the boundary between the external and internal toxidermas is not absolute.

3. In the determination of the *injurious agent*, it is essential not to overlook the following considerations: Among the various eruptions which may be caused by the same substances some are specific, plainly characteristic of the cause which has produced them (example: some bromide eruptions, some antipyrinides and arsenical keratosis); others are less characteristic and justify only a presumptive diagnosis (example: iodide acne, bromide acne); still others are commonplace, resulting from the action of many different kinds of substances (example: urticaria).

It is a matter of common observation that a given individual reacts to the same substance by an always identical eruption, without a proportion necessarily existing between the dose and the degree of the reaction. Frequently also the same individual reacts by the same eruption toward several different substances. It seems in such cases that the patient makes the eruption, the injurious substance merely playing an accidental determining part which elucidates and reveals, as it were, an existing pathological tendency. Its action has been likened to that of the fingers on the trigger of a loaded gun. It goes without saying that the artificial eruptions which appear under the influence of a variety of causes, are necessarily of an ordinary form.

Pathogenesis. — The pathogenesis of the toxidermas must be studied from the standpoint of: (1) The conditions under which the injurious action of certain substances is developed; (2) the mechanism of their action.

1. *CONDITIONS OF INJURY.*—The nature of a substance—its chemical properties—sometimes account for the action which it exerts upon the skin; this is true for the caustic agents, for substances having a strong reducing or oxidizing action, etc.

In other cases, substances are concerned which possess special biological properties and under these conditions one must essentially remain satisfied with the phrase: *Quia est in eis quædam virtus.* . . . The vesicants or rubefacients belong to this group.

Provided that the conditions of dosage, concentration and duration of action are present, the substances in this first class are sure to affect any human skin, without a predisposition of any kind being required. The resulting dermatitides are external toxidermas through direct contact and may be designated as *traumatic toxidermas* or *chemical dermatitides* (example: sulphuric acid, croton oil, etc.).

Other external or internal toxidermas are due to bodies whose chemical character and general biological properties do not explain this influence, which moreover is exerted only in an inconstant, accidental, even exceptional manner, so that one is compelled to attribute the abnormal reaction to conditions of territory, intolerance, or individual susceptibility.

As a matter of fact, there exists no sharp boundary line between the groups of substances acting on all persons alike (example: caustic potash), those acting on many persons (example: carbolic acid), or those acting only exceptionally (example: iodoform). However, in the presence of an inconstant effect, not explained by the conditions of quality, quantity and duration of action, one must necessarily suspect the intervention of a personal factor, a special susceptibility, or a peculiar capacity of reaction. This is what is meant by predisposition.

Predisposition and Idiosyncrasy.—The question of predisposition to disease, which is connected with that of immunity, has a scope far exceeding the limits of a book of this kind. The problem may be studied from a general point of view in connection with infections, intoxications, nutritional diseases, etc. I have frequently had occasion to allude to predisposition in other chapters, but as it plays the most evident part in the toxidermas, a few lines will here be devoted to its discussion.

In the first place, it must be emphasized, with Jadassohn, that the term predisposition is applied to two phenomena which up to a certain point are distinct:

1. Increased sensibility, or *hypersensibility,* causing a given individual to react to a minimum dose, greatly below that which is generally injurious, but the form of the reaction is not unusual. It has been stated that in case of hypersensibility, the question of

dosage of the injurious agent is of no importance, but this is obviously an exaggeration.

2. *Idiosyncratic intolerance,* in which an individual experiences injurious effects from a given substance which it would not produce in any dose on a normal individual.

It is not always possible to decide which of these phenomena is concerned in a special case; moreover, there exist varieties intermediate between these two forms of predisposition.

In current usage, the word predisposition is employed rather for a sensibility the reasons of which are suggested by a disturbance of the general health; when the cause of the intolerance is absolutely mysterious, one resorts to Greek and speaks of idiosyncrasy.

The real nature of so-called predisposition is unknown. Clinical experience teaches that it may be: general, the skin proving susceptible to all irritants; or special, intolerance existing only toward a single substance or group of substances; absolute or relative, namely more or less independent of the dose and the mode of introduction. A medicinal agent is sometimes tolerated when it is ingested or injected, but proves injurious on external application; this is common with mercury and the rule with iodoform, for instance. It is noteworthy that poisons when ingested reach the skin in a lower concentration, especially as the liver retains a considerable share.

Predisposition is congenital and sometimes even familial, or acquired. It is finally either permanent or temporary and may recur a number of times.

The *causes* of the intolerance are generally obscure, frequently multiple and inextricable.

Often a *general* disturbance must be held responsible, due to an infectious disease, physical or mental overstrain, a physiological phenomenon such as menstruation, pregnancy, the menopause, etc.

A *local* irritation or infection may invite a toxiderma; seborrhea and acne vulgaris are known to predispose to iodide, bromide and tar acne, just as dental caries and gingivitis provoke mercurial stomatitis.

Digestive disturbances, preëxisting or caused by the toxic agent itself, favor the development of toxidermas; changes in the gastrointestinal motility, secretions, fermentations, etc., may lead to absorption of abnormal products or to a reaction of these products on the medicinal substance. In the bromide and iodide eruptions, for instance, the role of digestive disturbances seems probable.

In other cases, hepatic inefficiency or renal insufficiency are held responsible, meaning a diminution in the antitoxic function of the liver or the excretory action of the kidneys; or functional disturbances of the cardiovascular apparatus, the nervous system, or the

glands of internal secretion may sensitize the skin or bring about one or another eruptive form and localization.

Idiosyncratic intolerance may subside in some cases under a gradual and continuous administration, with establishment of habituation or mithridatization. One may assume that the skin has acquired a greater resistance, or that elimination through the emunctories has improved, or that an immunization has occurred, of which the phenomenon of so-called anti-anaphylaxis is undoubtedly an example.

The opposite is noted in respect to certain bodies which are capable of *accumulation* in the organism. Digitalis, bromin, arsenic, chloral and even iodin, etc., are known to belong to this group. However, accumulation accounts only to a very limited degree for idiosyncrasy.

The usual course of events is as follows: An individual, apparently free from anomalies, is attacked, in consequence of the application or ingestion of some alimentary or medicinal substance, by an eruption of an erythematous, urticarial, eczematous or other type.

If he has never before absorbed this substance or come in contact with it, the case belongs simply to the class of idiosyncratic intolerance. Sometimes, however, he has previously tolerated it without disturbance. In the latter case, whether the toxidermic attack has occurred at the time of one of the disturbances mentioned above or in the absence of a demonstrable adjuvant factor it is almost the rule for this individual to be thereafter, for a fairly long or even indefinite period, hypersensitized to such a degree that the most minute dose of the injurious body, or perhaps of several others, will give rise to a fresh eruptive attack.

This acquired sensibility has very naturally been interpreted by some as a peculiar instance of *anaphylaxis*, to which we must devote a few words.

Anaphylaxis.—Ch. Richet discovered (in 1902) and gave this name to the special sensibility produced by the preliminary injection of certain toxalbumins; it differs from the accumulation of toxic agents in not manifesting itself immediately but only after a certain period of incubation. The conditions under which he observed this phenomenon and the principal facts concerning it are as follows:

Experimenting on dogs with the poison of actinia [sea-anenomes], Richet demonstrated that an animal injected with a sublethal dose will recover; a new similar dose injected a few days later remains without effect; but if at the end of two to three weeks the same dog is injected with an even ten or twenty times smaller dose, he will suddenly develop very serious or even fatal symptoms. These symptoms, the essential phenomenon of which is a sideration of

the nervous system with lowering of the arterial pressure, constitute the "anaphylactic shock." Richet assumes that the first injection has stimulated in the blood the formation of a toxogenin, which on contact with a new dose of its *antigen* combines with it to form a powerful poison, the *apotoxin*.

The experiments of Arthus, Theobald Smith and others have shown that this anaphylactogenetic power belongs not only to toxic agents, but that the injection of the blood-serum of an animal into an animal of another species will similarly sensitize it toward this serum. It is now known that in a general way all albuminoid substances will produce anaphylaxis.

In addition to this direct and "active" anaphylaxis, Richet discovered that there exists a "passive" anaphylaxis, in so far as an animal may be sensitized by injecting into it the serum of a sensitized animal. I have previously mentioned that the discovery of passive anaphylaxis has been utilized in proving the origin of certain urticarial eruptions, those due for instance to pork or mussels.

The term *anti-anaphylaxis* denotes the process by which the outbreak of an anaphylactic shock or crisis is prevented. Credit is due to Besredka for having shown that the preliminary injection of small doses of the determining serum permits the injection of the total dose one hour later without disturbance. A very slowly administered injection is in itself anaphylactic to a certain degree. I have pointed out that a rational attempt has been made to treat urticaria by anti-anaphylaxis. This therapeutic method will perhaps find numerous and important applications in the future.

That medicinal intolerance and anaphylaxis are not identical is apparent on the following grounds: In the first place, in spite of some contradictory experiments, it has not been established that non-colloid substances may produce an anaphylactic state. Furthermore, it is not uncommon for a toxiderma to manifest itself at the first use of a drug; in these cases, at any rate, there is nothing like the period of incubation which is required for the development of genuine anaphylaxis. Achard and Flandin were unable to produce in guinea-pigs either an active anaphylaxis with medicinal agents or a passive anaphylaxis by injecting them with the serum of patients suffering from eruptions due to antipyrin, iodoform, or quinine. Pending further research, medicinal intolerance must therefore be interpreted as a simple hypersensibility, not partaking of an anaphylactic character.

It is necessary to point out, however, that Ch. Richet showed in 1910 that animals which have been sensitized by an anaphylactogenetic substance are to a certain degree sensitized at the same time toward all poisons even crystalloids.

Looking at the question of idiosyncrasy from a wider perspec-

tive, one may well ask with him if, just as human beings differ in their psychic personality, they are not each endowed with a *humoral personality* peculiar to the individual; the personal differences in the humoral make-up, being due to heredity, ingesta, as well as the multiple intoxications and infections which they have experienced in the course of existence, accounting for the differences in the reactions toward injurious factors.

2. *PATHOGENIC MECHANISM.*—It is not necessary for the pathogenic mechanism of the toxidermas to be uniform and always identical; it is not even probable that this is the case.

The interpretation of external toxiderma is not difficult, the condition represents a sort of local chemical traumatism. It must be kept in mind, however, that in order to cause a reaction, the injurious substance must pass through the inert horny layer. The absorption of the normal skin is demonstrable only for volatile substances (ethereal oils, iodin, mercury, etc.), certain fatty substances and the keratolytic substances (salicylic acid). It has been shown, however, that potassium iodide, antipyrin and some alkaloids may penetrate the skin under certain conditions. Moreover, the slightest fissure of the horny layer constitutes a portal of entrance.

Many substances give rise to eruptions when absorbed by the external as well as by the internal route; one is led to assume that the injurious action is exerted as an idiosyncrasy on the constituents of the skin itself. It is certainly amazing to see to what degree the cutaneous sensibility must be augmented for a reaction to occur with the infinitesimal quantity of substance carried by the blood stream to a given square centimeter of skin; but one is prepared to admit this hypersensibility in view of what has been established in the external toxidermas.

It is very remarkable that this predisposition is often strictly localized and limited to a given territory which nothing seems to predetermine for this selection; the typical example of the antipyrinides, however, compels the recognition of this fact.

It has been supposed that the vasomotor or trophic (?) *nervous system*, damaged by the circulating poison, might intervene in the localization of the internal toxidermas, a metameric distribution of which has sometimes been reported. But the inflammatory character of the great majority of toxidermic lesions renders the idea of a purely nervous pathogenic influence rather improbable.

The role of the blood is indispensable in all not strictly local toxidermas; the injurious agent must be transported either to the nervous elements or to the skin. In a few exceptional cases, the blood medium is even morphologically altered; E. Hoffmann and

others have noted eosinophilia in hydrargyria and Leredde has demonstrated it in iodism.

The toxidermas were formerly very simply referred to *elimination*, or to an attempted elimination of the toxic agent by the cutaneous tissues. The purifying function thus attributed to the skin is now known to be in reality very limited, if not absent. It has not been demonstrated that in eruptions due to iodides or bromides, for instance, the altered skin contains a larger quantity of the metalloid than the neighboring skin. The positive findings of Adamkiewicz for iodin and of Gutman for bromin are offset by a mass of negative results; Pasini believes that in bromide eruptions the bromin exists in an organic combination not easily demonstrable. As to arsenic, it is known—and the fact has been confirmed by the careful analyses of Arm. Gautier—that the epidermis and its adnexa, hairs and nails, contain a proportion of the toxic agent relatively so high that the epidermic tissues must really be considered as normal routes of elimination of arsenic; but the condition exists both in the presence and absence of cutaneous lesions.

In certain toxidermas, the part played by microbic infections, especially through pyococci, is very evident. Thus the iodide, bromide and tar acnes, the eruption due to thapsia, the miliaria alba following mercury and the application of plasters, contain staphylococci with such regularity and abundance that the germs may be regarded as practically obligatory collaborators of the toxic agent.

The eczematous and bullous toxidermas likewise open the door to the same agents which may lead to generalization and persistence of an originally toxic eruption, the transportation being facilitated by scratching.

TOXIDERMAS FROM EXTERNAL CAUSES.

This section comprises different groups of cases, according as the eruption results from: (A) Badly tolerated medicinal applications, *external medicinal eruptions*. (B) Application of counter-irritants or caustics, *induced dermatitides*. (C) Substances used by the patient in his work, *occupational dermatitides*. (D) Vegetable or animal poisons, *dermatitides venenatæ*. (E) Applications made for the purpose of malingering, *simulated dermatitides*.

The morphological appearances assumed by these various toxidermas have been sufficiently described in the first portion of this book. They belong especially to the following groups: erythemas, urticaria, eczema, pustules, bullæ, ulcerations, gangrenes, dyschromias and folliculoses.

A. External Medicinal Eruptions.—The therapeutic substances which are capable of producing lesions or reactions that the physician had not expected, are innumerable.

Water, either pure or containing anodyne products, employed in prolonged baths, in moist dressings, etc., macerates the epidermis, thereby predisposing to pyococcal infections. The phenomenon of the "crop" (poussée), so common in the majority of watering places, is to a large extent referable to the water. Poultices act in the same way, particularly when the linseed has become rancid and therefore irritating. Excessive washing with soap and alkaline or sulphur baths may cause dryness of the skin, redness, desquamation and the formation of vesicles.

The majority of fatty or resinous substances which enter into the composition of *plasters* are capable, as a result of maceration, of irritating the epidermis and exciting eruptions accompanied by an abundant growth of pyococci.

Antiseptics, even the weakest, such as boric acid, sodium biborate, etc., may prove irritative to the skin. Some remedies of this group are entitled to special mention.

Pure *carbolic acid* is a caustic producing a white slough, almost without inflammatory reaction; strong or even weak improperly prepared solutions often give rise to *carbolic acid erythema*, sometimes to vesicle formation with edema, *carbolic acid eczema*, and even to gangrene when employed in dressings, a fact which must not be overlooked (Chapter XV).

Tincture of *arnica*, much liked by the laity for the dressing of contusions, has often a pronounced eczematizing action.

Formol hardens the epidermis, which cracks and peels off, under simultaneous production of a dermatitis which may be necrotic.

Salol, a compound of carbolic and salicylic acids, is like its components frequently the origin of persistent and progressive erythemato-vesicular dermatitis. As an admixture in tooth-washes or "catarrh powders," it gives rise to orbicular eczemas of the lips or nostrils which persist indefinitely when not traced to their real cause.

Iodoform is especially to be dreaded in cases of idiosyncrasy. Following its application in even trifling amounts, the neighboring skin becomes deeply reddened and covered with small confluent vesicles. This dermatitis is accompanied by an erysipeloid or pseudo-phlegmonous edema, especially on the face or on the genitals; it may even become generalized. The eruption terminates by desiccation or by suppuration; recovery takes place only after two or three weeks. It is certain that iodoform dermatitis results rather from the contact of the iodoform with the epidermis than with the cavity of an ulcer or a wound, or even with the surface of a mucous membrane.

General symptoms, delirium and even death, have also been observed and are referable to the absorption of this remedy.

The substitutes for iodoform, iodol, aristol, europhen, airol, etc., are less frequently injurious. *Orthoform* may excite an erysipeloid and gangrenous dermatitis.

Mercury and its salts, in external applications more often than after ingestion or injection, give rise in predisposed persons to eruptions known as *cutaneous hydrargyria*. It is true that the mechanical factor and the nature of the vehicle also play a role in mercurial inunctions; but the eruption frequently follows upon a simple application of blue ointment (Fig. 143) or dressings with sublimate, biniodide, cyanide, mercurial plaster and even applications of calomel.

FIG. 143.—Cutaneous hydrargyria of eight days' standing, occurring a few hours after inunction of the groins and the axillæ with blue ointment for phthiriasis.

The symptoms consist, in the first degree, of severe reddening with a burning or itching sensation, on which numerous miliary or submiliary vesico-pustules or small purpuric spots appear. This eruption, often localized in the groins, on the genital organs and in the axillæ, sometimes tends to become diffuse and then gives the picture of cutaneous hydrargyria due to internal causes. A more

or less dark and persistent pigmentation often follows the mercurial erythema.

Reducing agents, so widely employed in dermatotherapy, are all in varying degrees capable of producing dermatitis; this is accounted for by their special chemical effect, sometimes favored by an idiosyncrasy of the patient. Sulphur, sulphites, resorcin and naphthol are usually not injurious unless employed in excessive dosage. Pyrogallol stains the epidermis black; it may produce erythema with enormous swelling and even sloughing.

Chrysarobin, when dissolved by the alkaline sweat, gives rise to a brown or purplish, more or less extensive reddening with pruritus and sometimes a pseudo-phlegmonous infiltration lasting several weeks. The *bronzed erythema* of chrysarobin is typical. When applied near the eyes, the remedy causes a severe conjunctivitis with swelling of the eyelids, ulcerations of the cornea, etc.

The various *tars*, notably *oil of cade* may produce an ordinary erythemato-vesicular dermatitis. Their prolonged employment leads to hyperkeratosis. Some individuals moreover develop an eruption of folliculitis, solid papular elevations centered by a sort of brown comedo, with ultimate suppuration. This *tar acne* develops especially in hairy regions, particularly on the legs; it is obstinate and lasts several weeks (Fig. 123).

Oxidizing agents are relatively slightly injurious. However, *potassium permanganate* in strong solution or as a powder, very strong hydrogen peroxide and other *peroxides*, alter the horny layer and cause redness and vesiculation. *Picric acid*, recommended as harmless, is capable of producing erythema with edema and eczematous vesiculation, even when employed strictly according to prescription.

Dyes for the hair and beard usually have for their basis silver nitrate, subacetate of lead or pyrogallic acid; among their constituents, paraphenylene-diamin is especially dangerous. A few hours after the first application, or very often after a period of complete tolerance extending over months and years, an edematous and highly pruritic erythema suddenly makes its appearance on the upper part of the face and especially on the eyelids; rapidly extending to the entire face, the neck and sometimes the shoulders and the hands. If the reaction is severe, the erythema becomes covered with vesicles which rupture and are followed by oozing and crusts. The edema usually subsides in a few days, but the desquamation persists somewhat longer. An acute eczema of the regions indicated above, when the hair or beard has been artificially colored, will be readily traced to its true cause.

In order to prove that a particular substance has been injurious, a trace of the suspected substance may be applied on a very small

slightly abraded surface of the patient's skin and covered with gauze and a plaster; from twelve to twenty-four hours afterward an examination will disclose the presence or absence of a dermatitis. This "reaction test" thus strictly localized is devoid of danger.

The *treatment* of the external drug eruptions comprises in the first place the removal of the injurious substance, if traces of it persist. When possible, the employment of this substance and even of analogous products must thereafter be strictly avoided. Next, dressings appropriate to the degree and seat of the lesion may be applied, lotions, sprays, powders, soothing applications, as indicated in the treatment of erythemas, eczemas, etc.

Internal treatment with calcium salts or ichthyol has sometimes seemed to be of some value in my experience.

B. Induced Dermatitides.—In this paragraph will be considered the cutaneous irritations and lesions produced intentionally or otherwise by the physician, for therapeutic purposes. Skin lesions voluntarily produced by the patient with a view to malingering will be discussed separately (§E).

Everyone is familiar with the reactions caused by the customary counter-irritants, rubefacients and vesicants, such as: mustard plaster, hot water, chloroform, spirits of turpentine, ammonia, ethyl and methyl chlorides, tincture of iodin, iodized and analogous cotton, cantharides plaster or tincture, etc.

It is noteworthy that their application leaves in some individuals a very undesirable and persistent pigmentation; this excessive or prolonged action may cause sloughs, badly healing ulcers, etc., especially in children and persons with a delicate skin. Old iodin tincture contains hydriodic acid and becomes caustic. The vesico-pustules caused by thapsia, croton oil and tartrate of antimony are often followed by cicatrices, so that these substances should be employed very carefully.

As soon as the epidermis is damaged there is an open avenue for pyococcic infections and all aseptic precautions must therefore be taken before employing a strong counter-irritant; moreover, clean dressings must be applied after it has done its work.

Subcutaneous injections of camphorated oil sometimes and injections of paraffin for cosmetic purposes very frequently leave intradermic or hypodermic nodules which have been discussed elsewhere in this book (p. 276).

C. Occupational Dermatitides.—These comprise a great variety of eruptions ranging from acute erythema, or lichenoid thickening of the skin with cracks, to the different degrees of burns.

Eczema known as *occupational eczema* is especially common (Fig. 144), and *pyodermatitides,* in the form of impetigo, folliculitis, lymphangitis, etc., occur.

The seat of the occupational eruptions depends upon the region exposed to the injurious contacts.

The lesions therefore begin preëminently on the hands, more particularly their dorsal surface or still more often in the interdigital spaces; the nails in the prolonged forms are often worn, detached or grooved, fissured and dotted. Next, the wrists and forearms are invaded. The face and neck are affected primarily or secondarily. The covered parts, the scrotum, groins and axillæ are damaged in those cases where the clothes are impregnated with irritating dust, gases or fluids.

Fig. 144.—Occupatonal eczema of washerwomen.

The course varies according to the cause, form and degree of the eruption as well as the individual conditions in a given case. Occupational eczemas may become permanently established and recur incessantly, behaving as if the chemical traumatism had been only the determining cause of the onset of a diathetic eczema.

What has been said about the drug-eruptions permits me to be brief in regard to the eruptions of workers with chemical products (quinin, etc.), photographers (alkalies and reducing agents, amidophenol, metol, etc.) and operating surgeons; in the latter, the frequent scrubbing and washing with soap predispose to the injurious action of the various antiseptics.

Washerwomen, cooks and dishwashers usually have a smooth, shining, red and "parqueted" palmar skin; the excessive employment of potash soap, washing-soda and Javelle water, is here very apt to induce an artificial eczema with a topographical distribution on the hands and fingers, as stated above.

Bricklayers and plasterers have in the same regions a thickened sometimes fissured epidermis and are subject to eczematization of the fingers, hands and wrists, with pyodermatitis and lymphangitis; the handling of the various cements seems to be the essential cause.

Grocers handle a mass of irritative substances and sometimes present, especially in the cold season of the year, eczematiform or lichenoid lesions complicated by edema and fissures, which have been designated as "grocers' itch"; this term is fairly characteristic for the seat and gross appearance of this eruption.

Cabinet-makers, painters and workmen who use varnishes, essential oils, especially spirit of turpentine, are exposed to erythemato-vesicular and edematous eruptions bearing a close resemblance to acute eczema or dysidrosis as well as to chronic eczemas.

Sugar-refiners, confectioners, etc., who are in constant contact with sugar, often suffer from impetigos, whitlows and pyodermatitides.

There is finally an entire series of occupations in which the utilized materials themselves or their products give rise to a more or less ordinary dermatitis tending to secondary infections. It suffices to mention in this connection: the acneiform folliculitis, due to tar, petroleum and paraffin; the pyodermatitis of curriers, tanners and knackers; the various eruptions of mechanics, chauffeurs, etc., who are in contact with impure or rancid oils; the pruritus of bakers [bakers' itch], etc.

More specific in their cause and manifestations, are the occupational eruptions of workers in Provence cane;[1] spinners and weavers of flax; dressers of hemp; sorters or binders of wool; washers of silkworm cocoons; workers with chlorin (chlorin acne); and gardeners (poisonous plants), etc.

Pigeonneau, or rossignol[2] is an occupational disease of the hands in skin-dyers, which was carefully described by Brocq and Laubry. It is characterized by lenticular ulcerations, not very numerous as a rule, occupying the dorsal surface of the fingers or the hand; they are round or oval, covered with an adherent black imbedded crust; their border is raised and reddened; the ulceration is perpendicular and deep, with an irregular floor. The affection is very painful and heals rather slowly.

Treatment.—When dealing with an occupational dermatitis, the first thing to be done is to cleanse the parts by means of local baths or moist detergent dressings; these should be more or less frequently renewed and continued throughout the acute stage. In other cases the skin is cleansed with benzin, cold cream or vaselin. It is rarely necessary to resort to chemical neutralization or solvents, which are sometimes dangerous in themselves. Topical medication is governed as usual by the lesion (erythema, burn, eczematization or pyodermatitis) and not by the cause.

General treatment and especially hygienic rules must not be neglected.

[1] [A reed used for thatch-roofs.]

[2] [Young pigeon and nightingale respectively; popular names for this dermatosis.]

The difficulty in regard to occupational eruptions consists in the prevention of recurrences. Often, by utilizing precautionary measures and cleanliness, with protection of the exposed regions and attention to hygiene, the patient can resume his occupation and avoid the trouble which threatens him more seriously after the first attack. Sometimes, when there is an evident, well-marked and persistent predisposition, a change of employment or occupation becomes imperative. It is readily appreciated that the physician may find himself in a difficult position in cases of this kind.

D. Dermatitis Venenata.—Numerous indigenous or exotic *plants* are capable of producing by contact, not only a transitory urticaria like that caused for instance by nettles, but prolonged eruptions, especially of an erythemato-vesicular or bullous type, with edema, severe itching and sometimes fever.

The eruption appears soon after the contact; it may extend and become generalized. When the patient's occupation or the data furnished by him do not suggest the cause, one may mistake the etiology of the condition which may thereupon become obstinate and recurrent. A considerable number of seasonal eruptions of the hands and face are of plant origin; this must be kept in mind and inquiries in this direction must be made.

Among the poisonous plants which are most frequently responsible mention may be made of: daphne mezereum, several euphorbias, rhus toxicodendron (poison ivy) and several related species, primula obconica (primrose), and a few analogous hothouse plants, arnica montana, clematis, colchicum, scilla, thuya, some chrysanthemums, etc.

There also occurs occupational eruptions of vegetable origin, due to flax, cinchona, vanilla, bitter orange, satin [and other kinds of hard] wood, etc.

Many animals, of different species, not counting the parasites which will be discussed further on (Chapter XXIV) may give rise to eruptions through contact with them. Such are the medusæ, actinia, processional caterpillars [brown-tail moth], and a few others cantharides, etc.

Others have a poisonous sting, notably scorpions and many of the hymenoptera—bees, wasps, hornets, bumble-bees, etc. On being stung by one of these insects, an extremely severe pain is immediately felt and is promptly followed by urticarial redness with considerable edema; finally a bullous or vesicular eruption may develop, on the same spot or at a distance. A sting on the tongue or pharynx has been known to cause death. But aside from cases of this kind, alarming symptoms are sometimes observed, either on account of multiple stings or perhaps due to the effect of

direct penetration of the poison into a vein or to a special susceptibility. The venom of the hymenoptera, studied by Phisalix and by Calmette, is analogous to the venom of serpents. It may cause vertigo, vomiting, respiratory disturbance, weak pulse, fever, cold sweats, syncope and convulsions. Usually the disturbance passes off in a few days.

The *treatment* of eruptions due to contact with plants and animals consists in the removal of the cause and in dressing the lesions according to the type of eruption.

Against the stings of venomous insects may be recommended strong salt water, dilute ammonia, a solution of permanganate and rubbing with various fresh herbs, especially with parsley. In the first place it must be ascertained if the sting has remained in the wound and if this is the case, it must be extracted. Calmette has obtained excellent results from applications of calcium hypochlorite 1 to 60 or Javelle water 1 to 100.

E. Simulated Dermatitides [Feigned Eruptions].—These are observed among beggars, prisoners, soldiers and in hysterical individuals. Medicinal substances (see induced dermatitides), or poisonous plants, or sometimes burns, are especially utilized by malingerers for the purpose of producing eruptions intended to excite compassion, to avoid a tedious service, or to make the subject interesting. The procedure varies according to the intellectual standard of the individual and his opportunities for securing the necessary materials.

The lesions extend from a simple discoloration of the epidermis to eczematoid, erysipeloid and pemphigoid eruptions and even sloughing. Their configuration, arrangement and course naturally defy a general description. It is often their bizarre appearance that attracts attention; their angular or geometrical forms are apt to betray their origin; sometimes a trace of the material which has been used is discovered. It should be kept in mind that sycosis is relatively easily induced.

The seat of the lesions is always accessible to the hand of the patient. Inquiry and investigation frequently fail, the patient keeping up his denial in the face of the unmistakable evidence. Immovable occlusive dressings naturally suppress the pathological manifestations or cause them to shift their place. Unless the patient can be made to see the error of his ways and unless he consents to be cured, recurrences are apt to be indefinitely prolonged.

TOXIDERMAS FROM INTERNAL CAUSES.

These may be grouped under medicinal toxidermas, serum toxidermas, alimentary toxidermas and autotoxic eruptions.

A. Medicinal and Toxic Eruptions of Internal Origin.—It is impossible to make a complete list of all medicinal substances and poisons capable of producing eruptions. Some react rather frequently upon the skin; with others, this accessory effect is exceptional.

It is not so much the mode of absorption, the dose or the impurities in the remedy which must be held responsible as it is the idiosyncrasy of the subject.

The form of the eruption varies not only with the substance but also with the individual.

I shall restrict myself to a rapid enumeration of the ordinary eruptions and their most common causes and follow with a description of the more characteristic special eruptions.

Erythema in patches, *urticarial erythema* and *medicamentous roseola* represent the most common eruptions. The eruption is situated on the trunk, on the face and on the inner surface of the limbs; it is more or less abundant and extensive; it is sometimes scaly and often very pruritic. It may be accompanied by *enanthema*, conjunctival congestion, erythematous angina, but not by laryngo-tracheo-bronchitis as in measles. Fever and diarrhea are rare.

The eruption appears at a variable time, often very soon after absorption. It may develop in attacks or extend progressively; it attains its height in two or three days. It is not rare for the patches of a more or less urticarial erythema to spread after the fashion of an oil-spot while the center recovers or heals, resulting in *marginate* or *circinate* forms. In a few days, the eruption disappears without desquamation and without leaving maculæ.

Toxidermic erythemas are due especially to the following substances: quinin, antipyrine, morphin, the balsams, terpene, chloral, iodides, bromides, digitalis, salicylic, boric and benzoic acids, antimony, arsenic, exalgine, etc. In a soldier who had been exposed to the action of asphyxiating gases, I observed a marginate eruption of urticarial erythema, recurring for over eight months.

Scarlatinoid erythema is less common; it occupies especially the flanks, the great articular folds and may spread over the entire body, persist several days and be associated with a more or less marked and prolonged lamellar desquamation; the clinical picture will then be that of a primary acute erythroderma (Chapter VI). Recurrences are not uncommon. This form is observed especially after the administration of mercurials, quinin, chloral, opium, nux vomica, belladonna, salicylic acid, ipecac, antipyrin, sulfonal, benzoates, etc.

Urticaria (Chapter II), as common as the erythemas with which it is often combined, may be caused by the balsams, quinin, morphin, hyoscyamus, antipyrin, arsenic, iodides, bromides, chloral, santonin, etc.

Purpura is less frequent. A list of drugs which may cause its appearance has already been given (Chapter III).

Bullous eruptions sometimes result from the absorption of iodides, bromides, antipyrin, arsenic, quinin, salicylic acid, antimony aconite, etc. The appearance is that of a bullous erythema (Fig. 146), hydroa, or sometimes Duhring's disease.

The same substances may give rise to suppurative *folliculitides*, of acute or sluggish course.

Strictly speaking, there is no *eczema* of internal medicinal origin; but an already present eczema may become aggravated by any one of the above-mentioned substances, notably by the iodides, arsenicals, etc. In case of eczematosis, numerous medicinal agents, and also dietetic errors, etc., are capable of starting an attack.

Pigmentary spots sometimes originate under the internal use of antipyrin, arsenic, blistering gases and perhaps still other substances.

Gangrenes, in the form of gangrenous eruptions or gangrene of the extremities, have been observed after absorption of ergot of rye, carbon monoxide, antipyrin, arsenic, iodides and chloral.

Thallium salts produce a total *alopecia*.

Belladonna, morphin, cocain, nux vomica, aconite, etc., may cause *pruritus* without eruption.

It is obvious that the cutaneous manifestations due to internal medicaments are varied and often not characteristic; the same drug may produce very different effects. A suspicion of a drug-eruption is created by its behavior, its sudden appearance, its polymorphism, the absence of symptoms common in the diseases which it simulates and by its recurrence under similar conditions. An inquiry, which must sometimes be most minute and searching, will establish the correctness of the suspicion which has been aroused. Occasionally, if the patient wishes or consents, it is possible to make an experimental demonstration of the diagnosis by administering a small dose of the supposedly harmful drug. It has been said before (page 461) that it is not possible in these cases to establish a passive anaphylaxis in animals.

The following eruptions are less common; their characters suffice to suggest their origin; some are actually almost specific.

Balsamic Erythemas.—Copaiva, cubebs, santal and turpentine give rise to roseolar erythemas or erythematous patches, often urticarial and marginate, very pruritic, of a bright red color, occurring specially on the extensor surface of the large joints or on the upper part of the trunk, later on becoming more or less generalized. Their frequency has greatly declined since the less common use of the balsams in the treatment of gonorrhea.

Antipyrinides.—Antipyrin has already been mentioned among the substances capable of exciting various erythemas, urticaria,

purpura, etc. Very rarely, the eruption may simulate syphilitic roseola (A. Fournier), although it has a slightly deeper shade of red and is less persistent.

This remedy also produces persistent erythemato-pigmented patches, which were carefully studied by Brocq and are practically pathognomonic. These patches, single or scanty at the first onset, more numerous when the medication is continued (Fig. 145), are scattered irregularly and may be situated anywhere; they are round or oval, nummular or the size of the hand, dusky red, well outlined and slightly urticarial and cause a rather pronounced burning sensation.

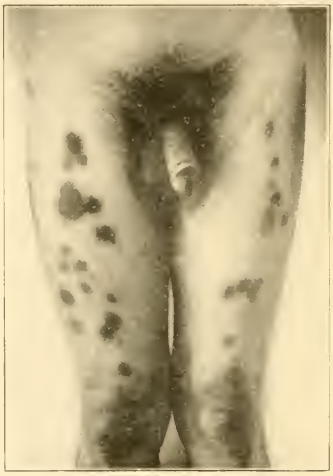


FIG. 145.—Antipyrinide. Remarkably profuse eruption of erythemato-pigmented spots.

After a few days the redness subsides and a fine or lammelar desquamation ensues; but the brown or even black pigmentation persists and fades only in the course of time. When the patient takes more antipyrin, after several hours or even in twenty minutes, the same spots again become congested and simultaneously new spots may appear, in haphazard distribution, taking a similar course. They are easily diagnosed from their circumscribed pigmentary and their stationary character.

Sometimes, a certain number of these spots or patches become the seat of *bullæ* or *vesicles*.

Cases of localized edema and fulminating gangrene due to antipyrin [and affecting the genitals particularly], have also been reported. Compounds containing antipyrin (such as "migrainin," etc.), of course, have the same effects. The erythemas produced by other analgesic and hypotonic agents are not so well known; fairly numerous but dissimilar observations have been recorded.

Iodides.—Potassium iodide and all other iodides or iodin compounds may give rise to diffuse erythemas, urticaria, purpura, or even to gangrene. *Nodosities* due to the iodides, analogous to those of erythema nodosum are likewise known to occur.

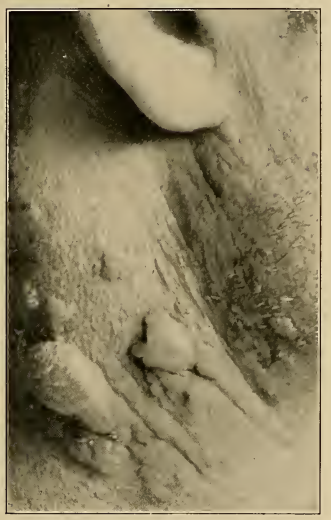


FIG. 146.—Iododerma bullosum of the neck, occurring a day after taking a mixture containing potassium iodide.

The most typical iodide or potassium-iodide eruption is *iodide pemphigus,* which has been mentioned before (p. 177) and would be better described, according to its appearance, as bullous (Fig. 146), ecthymatous, or fungoid iodide eruption. When the administration of the remedy is continued, the lesions become ulcero-vegetative, increase in size and often invade the mucous membranes; they are accompanied by general disturbances, diarrhea, albuminuria and cachexia and may cause actual mutilations or death itself.

Iodide acne is very common and develops almost exclusively in kerotic individuals; it affects the same areas as acne vulgaris,

from which it differs only in the more inflammatory character and the greater size of the papulo-pustules, their dark red color and their deep induration. Some lesions may be distinctly anthracoid (*iododerma tuberosum*).

Bromides.—The most specific eruptions are produced by potassium bromide, more frequently than by other bromin compounds usually after the large and prolonged doses prescribed, for instance, in epilepsy.

Bromide acne is like iodide acne; the ecthymatous and fungoid bromide eruptions resemble iodide eruptions of the same type.

Fungoid papulo-tubercular bromide eruptions are less common, but are pathognomonic (*bromoderma tuberosum*). It consists of a nummular or more extensive prominence, with a crusted mammillated or papillomatous surface, of a purplish red color and peculiarly soft quality, giving the sensation of wet velvet to the touch. The distinctly outlined patch is bordered by a margin of sub-epidermic suppuration; it enlarges by several millimeters daily and becomes confluent with neighboring lesions. The fungoid bromide eruptions occur chiefly on the face and especially the nose though often on the legs [most frequently in my experience] or the buttocks, but they may also be met with in other regions.

Their softness and rapid course distinguish them from papillomatous or fungoid tuberculosis, fungoid syphilides and pemphigus vegetans. Pasini interprets the cutaneous lesions as due to bromin liberated in the stomach under the influence of hypochlorhydria.

Hydrargyria.—Mercury and all its compounds without exception, whether absorbed by the gastric route, inhaled in form of mercurial vapors, injected into the mucous cavities, veins or subcutaneous tissues, may occasion an attack of cutaneous hydrargyria; infinitely small doses sufficing in the presence of a special idiosyncrasy. Hydrargyria through external applications is relatively frequent; through ingestion or injection it is very rare in proportion to the large number of patients to whom mercury is administered. It is advisable, however, to keep this possibility in mind.

Alley and Bazin have described three degrees of hydrargyria:

In the benign forms the trouble is limited to a localized redness with a few small vesicles, especially in the groins and on the inner surface of the thighs.

In the moderate form, there is a severe erythema occupying the large folds, the groins, the axillæ as well as the palmar and plantar regions; the red surface, sometimes dotted with hemorrhagic points, becomes covered with an abundant crop of small vesicopustules resembling miliaria alba. There is an intolerable itching and burning sensation; fever, digestive disturbances and albuminuria may be present. The crusts are followed by oozing, then by a more or

less persistent lamellar desquamation (see Fig. 143). The clinical picture may be exactly that of an acute or subacute primary erythroderma; upon occurrence of this eruption, hydrargyria should always be suspected.

The malignant form is accompanied by swelling of the face and the extremities, by large bullæ, pyodermatitides, abscesses, adenitis, angina and gangrene and may lead to death. Desquamation occurs in large shreds. It is noteworthy that in cases of cutaneous hydrargyria, mercurial stomatitis, colitis, etc., are not infrequently absent.

Arsenic.—Aside from the ulcerations of the skin and mucous membranes observed in occupational arsenic poisoning, the administration of arsenic as a remedy gives rise especially in the palmar and plantar regions, but also elsewhere, to erythemas, urticaria, purpura, bullæ, etc. The frequency of *zona* in patients who take arsenic has been noted. Injections of the arsenobenzols are followed, in rare instances, by more or less generalized eruptions of erythematous spots; even scarlatiniform erythemas have been reported.

Pigmentations, in spots or diffuse, as well as arsenical keratoses are more typical; I have seen the latter, associated with a dystrophic condition closely suggesting xeroderma pigmentosum or senile degeneration, give rise to multiple epitheliomas, the so-called arsenical cancer.

Treatment.—In the presence of a medicinal toxiderma of any kind, the essential point is the discovery of the injurious substance and the avoidance of its use. When its employment is absolutely required [as with arsenobenzol in syphilis] it should only be after a long interval, beginning cautiously with small doses, and [if possible] varying the form and mode of administration of the remedial agent. Rest, a laxative, abundant drinks, especially milk mixed with Vichy or Vals water and large enemas, may prove useful.

Some medicinal eruptions (bromides, arsenic, mercury) persist a really long time after the responsible medication has been stopped and one must be prepared for this. The employment of corrective compounds or of so-called antidotes is not to be recommended. The cutaneous lesions should be treated [symptomatically] according to the form of the eruption. Against the general symptoms of iodism, Milian has recently recommended adrenalin.

B. Serum Eruptions.—The disturbances which have been observed since the therapeutic employment of antitoxic sera (antidiphtheritic, antitetanic, antimeningococcic sera) were first attributed to the antitoxins, but are now known to be due to the vehicle, usually horse serum and may be produced by any foreign serum. The contributions of Arthus and von Pirquet, the clinical studies of Marfan, Weil-Hallé and Lemaire, among others, have helped to elucidate the relations existing between "serum sickness" and anaphylaxis.

True serum symptoms, namely those occurring after the first injection, are rather rare, being observed in only 14 per cent. of the cases, according to L. Martin. They may appear very soon, or more frequently after an incubation of ten to fifteen days and generally consist of urticaria or a marginate ephemeral erythema, preferably localized in the vicinity of the injection, or disseminated.

When another injection of serum is administered a long time (at least fifteen days, but even several years) after the first, the onset of anaphylactic symptoms is frequently seen (in 50 per cent. of the cases), developing either immediately or after four or five hours or sometimes after two to ten days.

The *symptoms* developing promptly consist of anxiety, dyspnea, weak pulse and vomiting; they are controlled by injections of adrenalin.

The more delayed disturbances manifest themselves under two types, which are often associated:

1. A local reaction, an erysipeloid or pseudo-phlegmonous redness with glandular swelling, lasting from two to four days. Marfan calls this the "Arthus phenomenon."

2. A general reaction, a more or less profuse eruption of urticaria, with swelling of the face, or a marginate erythema with extensive policyclic patches, accompanied by edema, sometimes by anemia, subicterus, purpura, general prostration, very painful arthralgias and a fever up to 38° or 39° C. [101°–102° F.], lasting twenty-four or forty-eight hours. These clinical symptoms are associated with somewhat variable hematological disturbances; leukopenia or leukocytosis, mononucleosis, presence of precipitins, sometimes of hemolysins, delayed coagulation, etc.

In order to avoid as far as possible the necessity for repeated injections in the course of a disease which requires serotherapeutic treatment, it is advisable to employ the serum freely and repeatedly within the first few days. But the fear of serum accidents should in no case cause the abandonment of a re-injection when this is demanded by the nature and course of the infection; this at least is the rule laid down by the most competent authorities.

All efforts should be made to avoid and control these symptoms. The best precaution consists in administering the re-injection very slowly, carefully watching the patient. Except in extremely urgent cases, it is even preferable to employ successive injections with small doses according to Besredka's method. Begin by slowly injecting 1 c.c. of serum under the skin; one hour later, inject 2 c.c.; finally, after another interval, the total amount of the dose is injected. Calcium chloride, in daily doses of 1 to 3 grams, also possesses, as shown by Netter, a distinct preventive action; it diminishes by three-fourths the number of eruption cases among

re-injected patients and has besides a curative value. Flandin, Martin and Darré, etc., have experimentally studied the serotherapy of serum symptoms endeavoring to produce an immunization by means of repeated injections of very minute doses, constituting a curative anti-anaphylaxis. Their results are inconstant.

C. Alimentary Eruptions.—In the case of an eruption occurring after the ingestion of a given food or drink, it is often very difficult to ascertain how far the symptoms are referable to the real toxicity of the suspected substance, the idiosyncrasy of the patient, or the auto-intoxications revealed on this occasion.

Any food or drink the ingestion of which is usually forbidden to persons suffering from urticaria, pruritus and eczema, for instance, may accidentally give rise to an acute eruption or increase a pre-existing dermatosis in predisposed individuals. (See Therapeutic notes, §12.)

The following may be considered as toxic, that is injurious, for most people: spoiled meats or fish, especially pork; very high game; certain meat extracts; spoiled or diseased mussels; edible mushrooms which have begun to putrefy, etc.

The eruption usually follows closely on the ingestion and lasts a very variable time. It consists of urticaria of all kinds, urticarial, scarlatinoid or polymorphous erythema, purpura and bullæ.

General disturbances, more or less pronounced or serious, are its usual accompaniments in the form of fever, vomiting, choleriform diarrhea and various nervous disturbances.

The *treatment* must aim primarily at emptying the digestive tract through all means at our command.

The patient must then be placed on a strict diet, or better a milk diet, for some time to come. The nervous symptoms are counteracted by symptomatic treatment and the same applies also to the cutaneous manifestations.

D. Autotoxic Eruptions.—Although these are not artificial eruptions, they may be considered as toxidermas and accordingly may be discussed in this chapter.

At the present day, a general description of the cutaneous manifestations of *arthritism* would be out of date; although some authors try to substitute chronic auto-intoxication for the vague term diathesis implied in this concept, the so-called *arthritides* of Bazin are now only of historical interest.

It is equally impracticable to draw a picture of the cutaneous manifestations of gout, uremia or urinary insufficiency, hepatic insufficiency, cholemia, the gastro-intestinal dyspepsias, etc.; an attempt of this kind would be blocked by too much that is unknown. In discussing the erythemas, urticarias, purpuras, eczemas, lichens, pruritus and prurigos, acnes, etc., what little is known of the relation

of these dermatoses to general nutritional disturbances has been mentioned. I repeat that eczematosis of adult or aged individuals may not infrequently lead to the discovery of a still latent internal cancer, an existing pyelonephritis or nephritis, etc.

It is only the eruptions of diabetics, the so-called "diabetides" of A. Fournier, that admit of a more or less general review.

Diabetic Eruptions.—These are of two kinds: Some might be considered as internal toxidermas, dependent solely on the altered organic environment; these include pruritus, chronic urticaria, eczema, purpura, gangrenes and xanthoma. Others require in addition the intervention of an external factor, such as the pyococcic infection in the case of impetigo, ecthyma, folliculitis, furuncles or carbuncle; irritation through the sugar in the urine, in the case of *genital diabetides*. The latter alone require discussion. They may constitute the first symptom to arouse attention and lead to the diagnosis of glycosuria. In the absence of the necessary cleanliness, especially in women, the sugar-containing urine on soiling the skin irritates the epidermis and creates an excellent culture-medium for the yeasts and the microbes of suppuration. These women will accordingly develop pruritus, chronic erythema, or an often very red, edematous and oozing eczema, with sharp sinuous outlines, marked by a raised horny border or a crop of pustules, with an acute, obstinate or relapsing course. This diabetic eczema of the vulva often extends to the thighs, the perineum, the groins and the abdomen. In diabetic men, the glans and especially the prepuce are often red, swollen, chapped and eroded; the preputial orifice finally presents a fibrous induration, with retraction and radiating fissures, or even with complete phimosis. These various lesions sometimes reveal the presence of diabetes mellitus.

In both sexes the clinical picture may become complicated by erysipelas or gangrene.

The *treatment* must aim at diminishing the glycosuria through diet, hygiene and if necessary by means of antipyrine or its analogues. Absolute cleanliness must be maintained; with frequent application of alkaline, weakly antiseptic or astringent lotions and bland powders. Painting with a 1 per cent. solution of silver nitrate is often useful. Circumcision should be avoided if it be possible, for it is not devoid of danger in these conditions. A "cure" at the Vichy [or other alkaline] springs is indicated.

CHAPTER XXIV.

NEURODERMATOSES; PRURITUS AND PRURIGO.

Pruritus, or itching, is a peculiar sensation which induces scratching; it is as incapable of definition as is a gustatory or a tactile sensation. It is irrelevant whether this sensation, in different cases, be compared to prickling, tickling, tingling, the crawling of an insect, etc.; the word *itching* includes all these varieties and is understood by everybody.

The finer pathogenesis of pruritus is entirely obscure. In order to interpret the dissociations of sensibility observed in certain diseases, physiologists have been led to suspect the existence of special organs, nerves or nerve-terminals, for the different forms of sensibility, some of which manifest themselves only on the skin and on the neighboring mucous membranes: touch, pressure, pain, heat and cold. It has been surmised that this may be true of pruritus also, but neither anatomy nor experimentation has confirmed this theory.

In all probability this mode of sensation is dependent on the ordinary sensory nerves, although it has been attributed by Jacquet to the sympathetic nerves.

Itching results in different cases either from a special property of the irritant: *artificial pruritus;* or from cutaneous lesions of various kinds: *secondary pruritus;* or from a special tendency of the individual, sometimes occasioned by local factors: *primary pruritus.* Frequently several of these conditions exist in combination.

The practically inevitable consequence of itching is scratching. When the latter leaves the skin intact or produces only very ordinary traumatic lesions, even when more or less complicated by secondary infections, the existing dermatosis is described as pruritus, adding to this term some qualification suggestive of the pathogenesis or clinical variety of the pruritus. However, when the scratching gives rise to certain special reactions in the skin (papules of strophulus, of prurigo, or lichenization), the dermatosis receives the name of *prurigo,* of which also several varieties are recognized.

The distinction between pruritus and prurigo rests accordingly, in my opinion, upon a morphological and directly demonstrable fact, namely the presence or absence of the peculiar papules which I have previously discussed (Chapter VII) or of lichenization. It is really of small importance, for it is not uncommon to find a pruritus becoming transformed into a prurigo after a certain time.

Under the special name of *neurodermatoses* are grouped pruritus and the prurigos which are neither artificial nor secondary, in which the itching appears as if primary and essential; it is dependent on a nervous disturbance of unknown character directly causative of the cutaneous manifestations.

Artificial Pruritus.—It is a matter of common knowledge that certain irritants of the skin possess pruritic properties in themselves. The crawling on the skin or the bites of parasites such as lice, bed-bugs, fleas, itch mites, oxyuris, the stings of the hairs of nettles or of the processional caterpillar, the application of a large number of substances of vegetable, animal or chemical origin give rise to more or less itching in all individuals.

It may therefore be stated, with Hebra, that under these conditions, pruritus is physiological.

Scratching in such cases may be really useful, being a defensive movement which may be reflex and sometimes unconscious, whose aim is the removal of the injurious agent.

Uncleanliness or, on the contrary, the excessive use of soap, etc., may also be the cause of persistent itching.

The severity and duration of artificial pruritus are governed by a personal coefficient; it varies with the age of the patient, his temperament, his physical and reactionary state, his hygienic habits and the possible existence of a faulty diathesis. It can be modified, moreover, by suggestion or autosuggestion; many people feel like scratching as soon as the conversation turns on lice or bugs. It may assume the proportions of an obsession, a neurosis or mania—a condition which is designated under the name of *parasitophobia, acarophobia,* or *dermatophobia.* Inversely, it is at least modified by habituation, intercurrent fibrile diseases, etc.

Secondary Pruritus.—Many dermatoses of external or internal origin include pruritus among their usual or possible symptoms.

When the pruritus accompanies or follows the eruption, the interpretation of the fact is relatively simple.

It may, however, precede it. In such cases, the question arises if the pre-eruptive pruritus is the effect of not yet perceptible histological lesions, or if it is a concomitant or preliminary effect of the cause which is about to give rise to the eruption, or finally if it is not itself the cause of this eruption by the scratching which it causes; in other words, if it is not a primary pruritus.

The solution of this question in the different cases which may present themselves, constitutes one of the principal difficulties of the pruritus and prurigo problem.

There would be no advantage in dwelling at length on pruritus in the dermatoses; this symptom, with its peculiarities, has been pointed out in each skin affection separately. The most pruritic

of these diseases are scabies, urticaria, eczema, certain medicinal eruptions, lichen planus, Duhring's disease, certain erythrodermas, some leukemias and mycosis fungoides.

As the result of scratching in the pruritic dermatoses the lesions of lichenization may appear and one may speak therefore of *secondary prurigo*.

On the contrary, many cutaneous affections practically never give rise to itching, notably the syphilides, psoriasis, lupus, leprosy, tumors, etc.

PRIMARY PRURITUS.

This designation is applied to pruritus which is not connected with an external and evident cause. The skin is healthy, but, nevertheless, there is itching, sometimes to an intolerable degree. Strictly speaking, this pruritus should not figure among the dermatoses; but although the seat of the disease is elsewhere, the dermatologist's advice is invariably sought because the symptom resides in the skin.

This primary pruritus may be diffuse and even generalized, or it may be localized; the fact must be emphasized from the start that an internal and general cause may manifest itself by a regional or partial disturbance.

Pathogenesis.—The pathogenesis of primary pruritus, also known as essential pruritus or pruritus from internal causes, has long taxed the ingenuity of physicians. Anticipating the data of positive knowledge, it has been assumed to depend sometimes on a systemic disorder through intoxication, auto-intoxication or nutritional disturbance: dyscratic pruritus; in other cases, on a nervous disturbance or even on a neurosis: nervous pruritus. The distinction between these two pathogenic mechanisms is sometimes not easily made and rests, moreover, merely on hypotheses.

Auto-intoxication actually seems to be involved in the pruritus of icterus, diabetes, uremia and dyspepsias, which have their counterparts in the toxic pruritus due to morphin, belladonna, caffein-poisoning, botulism, etc. However, in order to produce pruritus, the action of the poison must necessarily affect some part of the nervous system, terminal or central; making use of a bold figure of speech, it has even been said that it is the convolutions of his brain which the patient scratches on the skin. Hence, although of toxic origin, this dyscratic pruritus would be nervous in its mechanism.

On the other hand, it is an extremely obvious fact that a pruritus may result from a nervous perturbation, the proof is furnished by emotional pruritus, irritative pruritus, the pruritus of tabes, that of certain classified neuroses, such as neurasthenia, chorea, etc.

The statement has even been made that a pruritus not referable to an external cause or to an exogenic or endogenic toxic factor might be considered as an autonomous neurosis, an idea which is expressed in the term of neuroderma, introduced by Brocq.

By Jacquet, who made a specialty of the study of the sensory disturbances of the skin, pruritus was interpreted merely as the exaggeration of the normal cutaneous sensations; in the physiological condition, the total sensations coming from the integument constitute a harmonious whole, which may be called *enderma;* when this balance is disturbed, for example, under the influence of cerebral strain, or the absorption of certain foods, pruritus makes its appearance.

Etiology.—The causes of primary pruritus are infinitely variable and of different kinds. As *general and predisposing causes* may be mentioned the race, the geographical and social environment (J. C. White says that in the United States pruritus is a national disease; in European countries the Jews pay a heavy toll); a nervous or arthritic heredity, resulting from a bad hygiene of overnourished, gouty, diabetic, etc., progenitors; and the age of greatest activity, which is from twenty to forty years, in both sexes.

The following conditions act as favoring or determining factors: Brain-fag, which is very common in large cities; worry; late hours; grief; venereal excesses; cold and heat, some forms being seasonal; Duhring described a *pruritus hiemalis* (winter pruritus) which is often a prurigo; Bazin reported a pruritus *a calore* in persons employed in the vicinity of fires;—and hygrometric and barometric variations, to which some pruritics are susceptible.

To the same group of causes belong the following: Dietetic errors, overeating, abuse of meats, condiments, stimulating beverages, such as coffee, tea, alcoholic drinks, etc.; a bad condition of the teeth and gums, also habitual constipation, which act as intoxications; and the employment of certain remedies which cause a *toxic pruritus*, for example, belladonna, caffein, arsenic, morphin, opium, cocain, etc.

Diathetic or *dyscratic pruritus* is often referable to auto-intoxications, nutritional disturbances, functional disturbances or organic lesions of the viscera, the following being represented in this etiology: diabetes, gout, obesity, hepatism—with or without cholemia and often out of proportion to the degree of icterus, but perhaps related to the cholesterinemia, major and minor uremia, pyelonephritis, chronic enteritis, chronic appendicitis, circulatory disturbances, cancer, tuberculosis, the leukemias, etc.

The nervous disturbances accompanied by pruritus are less frequently those of organic origin like hemiplegia, brain tumors, general paralysis and tabes (Milian) than those of functional

character. *Nervous pruritus* is commonly observed among social failures, unhappy inventors, mental defectives, as the result of too much work or too much play and as the sequel of an emotional disturbance; the original depression is aggravated by the constantly recurring distress and by insomnia, leading to neurasthenia, melancholia and sometimes suicide. Formerly, cases of pruritus connected with pregnancy, menstrual disturbances, utero-ovarian, prostatic or vesical diseases, were interpreted as reflex pruritus, but these are now referred rather to auto-intoxications.

Hematogenic pruritus is observed, although inconstantly, in the leukemias and may lead to the recognition of the blood disease.

Determining causes are those which may excite a latent pruritus and bring on a crisis. The time of disrobing at night is very commonly dreaded by pruritic patients, as it becomes the occasion of a real paroxysm; it is impossible to say if the exposure to cold, contact with the air or decompression are responsible; at any rate, many healthy persons scratch when disrobing, notably the majority of women after having removed their corsets, belts or garters.

The attacks may also be brought on by a bath, rapid walking, cold or heat, even by a perfectly harmless meal. Certain persons are attacked by pruritus immediately after the ingestion of some food or drink, before any actual absorption can have taken place as if under the effect of a reflex; as particularly harmful in this respect are mentioned acid fruits, mussels and shellfish, cheeses, spices, alcoholic beverages, tea, coffee, etc.

Often, however, the attacks occur spontaneously, even during sleep, apparently resulting from a nervous discharge.

A few general data, well elucidated by Jacquet, dominate the etiology of pruritus:

1. Predisposing and determining causes, internal and external, are often associated and act through a cumulative effect constituting the *prurigenetic summation.*

2. A preceding pruritus invites another; apparently each organic cell stores up more or less the stimulation which it has undergone, through a kind of local memory; this is *prurigenetic mnemoderma.*

3. The accumulated sensory energy may become stationary or it may, on the contrary, become transported and transformed, representing *sensory metastasis.*

Symptoms.—Pruritus, being an essentially subjective phenomenon, manifests itself only by scratching. Just as there are all possible degrees of itching, from a slight distress, which is easily forgotten, to an imperious, inexorable necessity, against which no provision holds out, so there are likewise all sorts and forms of scratching. An insignificant pruritus yields to simple rubbing with the finger-tip; a severe attack requires forcible scratching with the

nails or rubbing with a rough towel, a brush or some other instrument which the patient uses as a curry-comb; sometimes a cold or hot application may accomplish the same object.

No relation, however, can be established between the severity of the pruritus and the scratching, which is modified by individual factors or conditions inherent to the disease.

Whoever has witnessed a characteristic pruritic crisis will retain a lasting impression. At the onset, the patient endeavors to control himself; gradually he yields to the need of scratching, which incessantly increases, its satisfaction being accompanied by a truly voluptuous sensation; all restraint is promptly lost; pale, anxious, distracted by his trouble, the patient furiously excoriates himself, mutilating his skin and literally torturing himself, as if in the power of a blind force. Sometimes it is not until the skin is raw and streams with blood that relaxation occurs, satisfaction is obtained and the attack is over. The patient remains exhausted and as if ashamed of himself. Comparison with an epileptic attack and that involved in the term of "cutaneous masturbation" is entirely justified.

It is impossible to say why scratching, even when carried to such an excess, produces relief; but all pruritic patients declare that the burning pain of the excoriations is preferable to the annoyance of the itching.

The attacks usually last from five to fifteen minutes, sometimes an hour or longer; there is no rule as to their frequency and the rhythm of their recurrence.

The pruritus described above may be diffuse and even generalized; it is more frequently partial, regional, localized on a more or less extensive portion of the integument. But a diffuse pruritus sometimes tends to become localized after a certain time or,inversely, a regional pruritus may be seen later on to radiate and become diffuse.

It is a noteworthy fact that a localized pruritus, originating under the influence of a local cause, may recur *in situ* on the occasion of an internal or general cause, through the effort of the mnemoderma referred to above; in this way an anal pruritus, for instance, caused by oxyuris or hemorrhoids, will recur at the time of dietetic errors or overexertion.

The consequences of scratching are partly immediate and partly remote. The traumatism produces in the first place congestion, with local heat and more or less interstitial exudation, that is, simple or urticarial erythema.

I believe that by itself alone it may also excite an eczematous reaction, traumatic eczema (Chapter IV).

More forcible scratching causes linear excoriations, usually directed in the course of the nails, which suggests their origin in spite of denial on the patient's part. Often the excoriations are punctiform and occupy follicular prominences, because from the beginning of the irritation the congested follicles have been raised by a spasm of their erector muscles and have projected above the general level. These excoriated follicular papules, covered with a bloody or serous crust, must not be confused with the papules of prurigo.

The remote effects of scratching are in part of a special kind, consisting of papulation or lichenization and suffice to place a given case of pruritus in the group of the prurigos.

Others are of an ordinary kind, namely secondary infection by pyococci, through the avenue of the traumatism giving rise to the most varied pyodermatitides. Even in the absence of suppuration, chronic scratching causes hypertrophy of the concatenated glands. Pigmentation, more or less diffuse, is likewise common. The worn-off nails which become polished and shining, have been mentioned elsewhere (Fig. 138).

It will be seen, with Jadassohn, that from the standpoint of the effects of scratching, the pruritic diseases may be divided into two classes: in the first, the patient's nails tear off everything that protrudes and even dig into the epidermis to tear off shreds of tissue; this *biopsic pruritus*, as it was called by Besnier, is observed in scabies, pediculosis, sometimes in diabetes, Duhring's disease and the prurigo of Hebra. Pyodermatitides are commonly present in these cases.

The second form of pruritus, although sufficient to cause insomnia, is relieved by friction, pressure, etc., and is unaccompanied by any traces of scratching. Senile pruritus, the pruritus of lichen planus, of some types of icterus, of urticaria and of phthiriasis, belong to this group. The reasons for this difference are unknown.

Clinical Forms.—It is not easy to formulate a picture of the forms of pruritus according to their origin. The following general remarks may be made on this subject.

Dyscratic pruritus, that of diabetes and icterus in particular, is often generalized, is observed in relatively aged individuals, is especially nocturnal and gives rise to deep multiple excoriations.

Nervous pruritus, or *neuroderma* of Brocq, attacks youthful or adult persons, is often regional or partial, occupying for example the large articular folds, the external aspect of the limbs, etc. It has a marked tendency to lichenization and consequently to prurigo vulgaris.

Hematogenous pruritus is accompanied by a change in the blood (leukemias and anemias), easily recognized when kept in mind and looked for.

Senile pruritus, described by Willan, formerly covered almost all the forms of pruritus of aged individuals. At the present time this name is restricted to a chronic, almost invariably generalized, remittent pruritus, in which the skin is flabby, dry, rough or smooth, more or less senile, but very resistant to scratching. As a matter of fact, even in severe senile pruritus, neither excoriations, nor urticaria, nor papules, nor lichenization are noted. This form is very rebellious to treatment.

Localized pruritus is provoked by and depends upon either a local or a general cause. A general prurigenetic cause often localizes its effects in a region which has first been irritated in some way or takes advantage of an ordinary local lesion, such as varicose veins or metritis, to lodge in its vicinity. The interest in this localized pruritus is derived from the existence of these *predisposing conditions* which it reveals and which can often be remedied. Not infrequently it becomes eczematized, thereby leading to confusion with the pruriginous eczemas, or it may also develop into prurigo.

Anal pruritus, which is perhaps the most common variety, at any rate one of the most obstinate and demoralizing forms, is often connected with the presence of oxyuris, lumbricoides, hemorrhoids, fissures, or with habitual constipation.

Perigenital pruritus, sometimes associated with the preceding, is frequently related to diabetes, cystitis, diseases of the prostate, gonorrhea, or strictures of the urethra. *Vulvar pruritus* is suggestive of leukorrhea, diseases of the vagina, uterus and adnexa; it is not uncommon at the menopause or after spaying. It may create the suspicion of or excite masturbation.

Nasal and *peribuccal pruritus* is often caused by coryza, rhinopharyngeal lesions, dental caries, neglected inflamed gums, or badly fitting artificial dentures. *Buccal pruritus* may occupy the lips, the cheeks, as well as the tongue; it is often associated with the neuralgia known as *glossodynia.*

Pruritus of the hairy regions is usually symptomatic of parasites or of kerosis, pityriasis, etc.

Palmar and *plantar pruritus,* described by Alibert and Hebra, is always symmetrical. It is rare and is encountered in unbalanced and toxic persons. It often assumes the form of burning sensations with nocturnal paroxysms. There is no trace of scratching. It must be distinguished from dysidrosis and from eczema.

PRURIGO.

I employ the term prurigo to indicate that group of pruritic affections in which the itching, which is the primary phenomenon, becomes associated under the influence of scratching with special

cutaneous reactions, in the form of lichenization and prurigo papules.

This definition, which is not given by all authors, is justified by the historical development of this question and meets with the nosographical requirements.

Willan grouped prurigo under his heading of papules, which comprised: (1) strophulus; (2) lichen; (3) prurigo. Credit is due to Cazenave and Canuet for recognizing that in prurigo the itching is primary and the papule secondary. The elder Hebra again reverted to the idea that prurigo is characterized by a primary papule, a view which has since been refuted. Modern investigations, especially those of Brocq and Jacquet, have established the almost universally accepted fact that the papules of prurigo and lichenization are secondary to the pruritus and due to the scratching.

Accepting the above definition (prurigo = primary pruritus + special papules or lichenization), the diagnosis of the prurigos becomes easy and the relations which they bear to associated dermatoses are readily understood.

In *pruritus*, there may be traumatic lesions from scratching, pyodermatitides and artificial eczema, but there are neither lichenization nor prurigo papules.

Urticaria is a syndrome in which pruritus and scratching are accompanied by the rapid appearance of very ephemeral urticarial papules. In some forms of prurigo the skin is seen to react in the form of urticaria, especially at the onset; the etiology of the chronic urticarias coincides in its general outlines with that of pruritus and the prurigos; we must therefore admit the existence of a certain kinship between these two groups of dermatoses.

Eczema, irrespective of its cause, may assume a lichenoid appearance and become extremely pruriginous. On the other hand, the prurigos frequently become eczematized. It is readily understood that the differential diagnosis between these two groups of cases, lichenoid eczema and eczematized prurigo, resting as it does merely on the priority of the pruritus or the eruption and on the more or less diffused lichenoid state, often presents almost insurmountable difficulties.

Several other pruritic dermatoses may ultimately become associated with lichenization. The existence of *secondary prurigos* may accordingly be admitted, as well as the more frequent occurrence of secondary pruritus.

Symptoms.—It is not necessary to repeat what has been said about primary pruritus, its etiology, its pathogenesis and its clinical manifestations.

The papules of strophulus and of prurigo have been already

described (p. 140). All that remains to be discussed in this connection is lichenization.

Lichenization (Besnier), which was first fully described by Brocq under the name of lichenification, is a chronic more or less persistent change in the appearance and the structure of the integument (Fig. 147). The lichenized skin is thickened as a whole; it may be described as striated, wrinkled, leathery, without either of these words conveying an exact idea of the condition. Its peculiar appearance is really characterized as an exaggeration of the fine

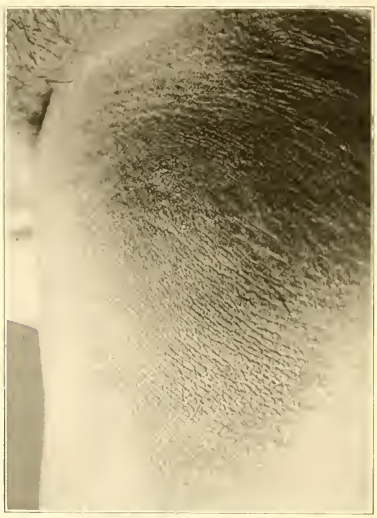


Fig. 147.—Lichenization of the skin of the internal aspect of the thigh, in a case of prurigo vulgaris, in a woman aged forty years.

striæ which normally traverse it, which results in a criss-cross network with fairly regular meshes, more or less wide or narrow according to the region affected, resembling the cross-hatching of an engraving. The meshes are square, lozenge-shaped, or polygonal; they have a flat surface and usually present the appearance of smooth shining facets, like mosaic work. Sometimes they are covered with fine scales.

The lichenized skin is less supple than the normal skin; its color is normal, or more often of a grayish or brownish hue; sometimes it is hypochromic.

Its histological structure is less altered than one might be led to believe; the lesions consist of acanthosis, with elongation of the papillæ and moderate infiltration of the papillary body.

The lichenization occupies surfaces of very variable extent, in patches or areas, with diffuse borders, where there is a gradual transition to a normal appearance. [It is characteristic of the border that it is not limited by curved lines but is made up rather of short rectilinear elements.] On these borders, or on a surface in course of lichenization, only a few shining polygonal facets are noted, appearing very slightly papular; but not indurated.

Lichenization must be distinguished from: the patches of lichen planus (Fig. 33) which are made up by the confluence of genuine planus papules and surrounded by typical papules; and from the lichenoid condition sometimes assumed by eczemas, eczematides and psoriasis; this is characterized by thickening and accentuation of the folds and furrows, but it is red, without shining facets and distinctly circumscribed at its borders. The striated and roughened condition sometimes presented by the skin of the genitocrural regions in gonorrheic women, a condition described by Brocq and L. Bernard, very closely resembles lichenization, although the surface according to these writers, is more villous and velvety.

Lichenization may be primary and pure, that is, it develops on a previously healthy skin under the influence of rubbing and scratching caused by pruritus; under this form it is the principal and most characteristic lesion of diffuse prurigo vulgaris; or it may be associated with the above-mentioned dermatoses, lichen, eczema, eczematides, etc.

Since lichenization is a peculiar reaction of the skin under the influence of repeated traumatisms, the question may arise why does it not develop on all surfaces exposed to friction, or in all pruritic persons who scratch themselves. I shall restrict myself to giving the explanations offered by Brocq, who says that some cutaneous affections modify the vitality or the nutrition of the tissues in such a way that lichenification occurs with the greatest facility, whereas in other pruritic affections the resistance of the tissues seems to be normal or even increased; moreover, some individuals are especially predisposed to react in one or another way. [It must be admitted that this explanation explains nothing; it simply restates the facts.] The same theories are applicable to the pathogenesis of prurigo papules.

As to the papules of strophulus, the question of their being of primary origin or secondary to the scratching, remains doubtful; it seems to me that they are often encountered at points of the skin which the patient can in no way injure or scratch.

Clinical Forms.—The prurigos constitute a continuous series of morbid types, extending from cases related to urticaria to formidable dermatoses such as prurigo ferox. It would be desirable to classify them into varieties and this has been attempted by many dermatologists, who always emphasized those types which they had been best enabled to study. Hence the subdivisions of some do not agree with those of others and it is very difficult to find one's way. Without aiming at completeness, I shall limit myself to describing three principal forms:

Acute Prurigo Simplex or Strophulus.—The affection named strophulus by Willan and Bateman and *acute lichen simplex* by Vidal, was given by Brocq the excellent name of *prurigo simplex acutus*, a name which has been received with general favor; abroad the terms of *urticaria papulosa* and *lichen urticatus* are often employed.

The condition is an acute benign prurigo, characterized by a special and exclusive lesion, the strophulus papule, generally originating upon an urticarial base; an eruption advancing in rapid, successive or overlapping crops, scattered over the entire body; the usual absence of lichenization and eczematization; a limited duration, of a few weeks to a few months, but with possible relapses; and on the whole a favorable prognosis.

Etiology.—Strophulus is of extreme frequency in early childhood; later in life it is observed much more rarely, but is not unknown, however, in the period from fifteen to twenty-five years.

A predisposition in certain families is obvious. It is encountered in apparently perfectly healthy children. However, two determining etiological factors can often be recognized: (1) Overfeeding or underfeeding, digestive disturbances, gastro-intestinal fermentations, constipation, etc.; (2) the influence of teething, which is accompanied by nervous disturbances, excitement, insomnia and often also by digestive disturbances. I have elsewhere pointed out that the second period of life where strophulus is frequently met with corresponds to that of the eruption of the wisdom teeth.

Symptoms.—Suddenly, in the midst of health, or sometimes after a slightly febrile general malaise, lasting one or several days, the eruption makes its appearance. It consists of urticarial spots surrounding and at first concealing the *papule* which has been previously described. Sometimes, a few urticarial elevations without papules are likewise noted and are easily brought on by scratching.

This eruption is located anywhere, at the onset preferably on the upper extremities and on the trunk; later, on the lower limbs, on the neck and on the face; the palms and the soles are only rarely involved.

An eruption proceeds in crops of four or five, to about twenty lesions; it recurs every day, or every second or third day. The urticarial spot lasts only a few hours and then it fades, but the papule lasts from four to ten days. It is therefore not uncommon to see children covered with lesions of different ages (Fig. 148), some incipient, others at their acme or undergoing retrogression; and sometimes with not very persistent macules.

Sometimes the lesions are small vesicles originating on an urticarial base; on the palms or the soles these vesicles may attain the size of a lentil.

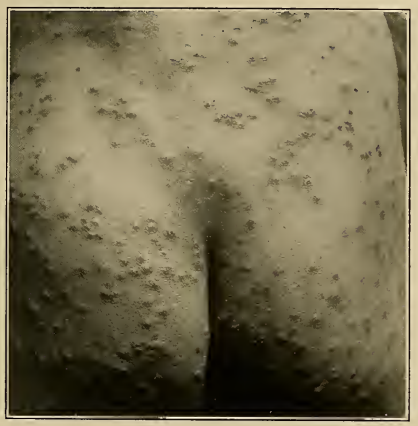


Fig. 148.—Profuse eruption of strophulus, or prurigo simplex acutus, in a boy aged six years.

The course extends over a very variable period, from three weeks to three months; recurrences are common in little children; toward the age of three years the disease usually subsides. If it persists, there is danger of the development of Hebra's prurigo.

Pruritus is variable and remittent, but often very severe. Some individuals scratch furiously and excoriate some of their papules; it seems, although this cannot be positively stated, as if the scratching gave rise to new papules. Eczematization and pyodermatitis are rare.

The *diagnosis* rests upon the demonstration of the typical papule, capped by a tiny lenticular yellow crust. In infants strophulus is often confused with urticaria, the stings of insects, sudoral and medicinal eruptions; in the young, with acne, papular erythema,

acute disseminated eczema. Cases where the eruption is plainly vesicular may suggest varicella; it should be kept in mind that the lesions of the latter are less pruriginous [are most abundant on the back and frequent on the scalp] and do not spare the buccal mucosa.

Prurigo simplex is evidently insufficiently known, for it is constantly being re-described by physicians under some arbitrary denomination.

The *treatment* consists in correcting and regulating the diet; it is often advisable to diminish the quantity of milk and give a purgative or laxatives, among which calomel is most to be recommended for children.

Locally, acidulated, carbolized or anodyne washes are preferable to baths, which are often not well borne. Zinc pastes with tumenol and tar and occlusion by well-applied bandages when possible, are effective against the itching.

Hebra's Prurigo.—From the chaos of the pruriginous diseases, Hebra picked out a morbid type which is designated under his name; a mild form (*prurigo mitis*) is known, as well as a grave and chronic form, characterized by a special course and by very polymorphous and severe lesions. It corresponds to the lichen agrius of the ancients and more or less accurately to the lichen polymorphe ferox of Vidal.

As a rule the disease begins in the course of the first year of life, in the form of urticaria or strophulus, with severe, recurrent, very pruriginous attacks, but in many cases the pruritus comes on directly, without premonitory eruptions.

At the end of a year or two, it reaches its fully developed stage and the clinical picture is characteristic: The child is tormented by incessant itching, or the pruritus may vary according to the season of the year. The skin is covered with linear or papulofollicular excoriations, crusts, cicatrices, diffuse or regional eczematizations, pyodermatitides; it is thickened, coarse, pigmented and lichenized over large surfaces. The external aspects of the extremities are chiefly affected, less often the trunk and sometimes the face; the large articular folds almost invariably escape. From time to time, more or less large prurigo papules are encountered, intermingled with the lichenization.

In the least scratched regions, the skin has an earthy color and often resembles goose-flesh; the prominence of the follicles is attributed to contraction of the erector muscles. The hairs, although abundant at first, soon become worn off and disappear.

The lymphatic glands of the groins and axillæ are always enlarged, and their enlargement is often very evident. Eosinophiles are usually present in large numbers in the blood and in the skin itself.

The little patients are sickly, irritable, timid, their persistent sufferings and habitual insomnia accounting for their gloomy disposition. When they grow up, their infirmity condemns them to a solitary existence; they can share neither in amusements nor in attendance at school.

The *course* of Hebra's prurigo is remittent; periods of relative calm are followed by aggravated attacks lasting several months. It is usually not until puberty or even about the age of twenty to twenty-five years that the disease subsides and becomes attenuated; from this time on, without knowing the patient's history, one might mistake it for a chronic eczema or a prurigo vulgaris. The pathological manifestations vanish as a rule in mature life or old age, if the patient live long enough.

In the *etiology*, the hereditary conditions mentioned above rank first in order, perhaps also to some extent errors in feeding in early childhood. Like other observers, I have several times noted the coincidence of Hebra's prurigo with asthma. [The possibility of sensitization to a particular article or kind of alimentary substance (*e. g.*, milk, eggs) must be considered in the etiology.]

Several varieties of this prurigo may be described:

In the *Hebra-Kaposi type*, the cutaneous lesions increase in severity from the head to the legs.

In what has been called the French type, they predominate on the face and the upper extremities, being on the contrary less well marked on the legs and on the trunk.

Prurigo ferox (polymorphous lichen ferox of Vidal) is a rare type, characterized by prurigo papules scattered or grouped on the extremities (Fig. 149), the body and even the face; they are of considerable size, approaching the size of a cherry-pit or half a hazel-nut, often excoriated or vesicular at their apex. On the other hand, lichenization, while constant, is less pronounced. The itching is intense and the disease is extremely obstinate and of indefinite duration.

[*Prurigo nodularis* (Hyde) is closely related to the above. All the cases described in America have developed in middle-aged women. The lesion, five to twenty-five in number, located almost exclusively on the extremities, are pea to hazel-nut in size, discrete, often capped by small vesicles, intensely pruritic and rebellious to treatment. The surrounding skin is normal in appearance.]

Cases of Hebra's *prurigo of late onset* may possibly occur; some are of doubtful nature and liable to confusion with severe cases of prurigo vulgaris; others are not infrequently referable to the leukemias, especially to pseudo-leukemia with polynucleosis (*prurigo lymphadénique* of Dubreuilh).

Prurigo Vulgaris.—Under this name I designate the common form of prurigo, namely the group of cases which belong neither to simple pruritus nor to acute prurigo simplex nor to Hebra's prurigo.

A distinction is made between: (1) A diffuse or generalized form; it corresponds in a considerable number of cases to the diathetic prurigos of Besnier, to the diffuse neurodermatitis of Brocq, which he has more recently designated as "diffuse pruritus with lichenification." (2) A circumscribed form, corresponding to the circumscribed lichens of the older authors, to the chronic lichen simplex of Vidal, to the papular eczemas of Hebra and his school, to the dermatitis lichenoides pruriens of Neisser, to the circumscribed neurodermatitis or circumscribed pruritus with lichenification of Brocq.

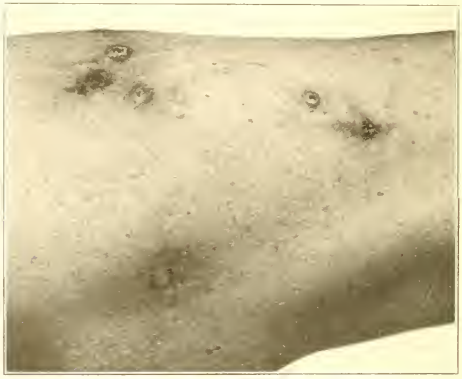


FIG. 149.—Prurigo ferox with large papules (polymorphous lichen ferox of Vidal.)

Prurigo vulgaris is characterized by primary pruritus; polymorphous cutaneous lesions among which lichenization and eczematization predominate; and by a generally subacute remittent course.

1. *Prurigo Vulgaris Diffusa.*—Its etiology is that of the primary prurigos in general, hereditary factors, neurasthenia, metabolic and nervous disturbances, abuse of coffee or alcohol, emotional causes, all of these play an obvious role, as well as the diathetic nutritional disturbances which are classified as "arthritism."

It may begin in childhood, but especially between twenty and thirty years; it is less common after fifty.

The affection appears abruptly, often after an emotional shock, persisting for a few weeks; then follow periods of subsidence and intermittent recurrences, sometimes of seasonal type.

During the attacks the itching is continuous, with evening and irregularly periodical crises; the scratching may at first give rise to urticaria or erythema; but more or less rapidly, in different individuals, sometimes in a few days, it leads to the production of indistinctly outlined surface lichenizations, symmetrically occupying the four extremities, the thorax and the flanks and sometimes the face, which becomes dull and grayish, with worn-off eyebrows. The combination of this diffuse lichenization with medium-sized and indistinct papules, with pigmentation, with traumatic eczema, excoriations and pyodermatitis, constitutes a picture characteristic because of its diversity (Fig. 150).

The disease is of indefinite durations covering months or years; it may heal and is often replaced by asthma, hay fever, bronchitis, enteritis, etc.

Fig. 150.—Diffuse prurigo vulgaris; right forearm of a young woman, aged twenty-three years, whose four extremities presented similar lesions; these lesions consist of diffuse lichenization, with pigmentation and numerous excoriated papules (diffuse neurodermatitis consisting of pure lichenization, Brocq).

There are numerous *varieties:* Cases occur in which the prurigo follows upon a long period of simple pruritus; or an eczema may occupy the foreground, more or less concealing the other signs, so that many of these patients pass under the label of chronic eczema; or the small and flat papules may lead to a confusion of prurigo with lichen planus; and finally, there are intermediate cases, with but few foci between the diffuse and the circumscribed form.

Under the name of *prurigo hiemalis,* a variety of this prurigo described by Duhring is designated, more common in North America, especially in men, characterized by its evident relation to the cold season. The pruritus appears every fall, is more severe in cold weather and ceases in the spring. The first attacks frequently date back to childhood. The crises are vesperal and nocturnal, or may occur in the daytime under the action of heat,

The most common site of the itching is on the legs, the thighs and sometimes the upper limbs. The condition is a simple pruritus in some persons, but in the majority of cases becomes a prurigo.

J. Hutchinson has described a *summer prurigo* in every way comparable to the preceding.

2. *Prurigo Circumscripta* or *Lichen Simplex Chronicus of Vidal.*—The etiology is the same as in the diffuse form; sometimes there exists a localizing cause, traumatism, internal lesion of the vicinity, etc. Circumscribed prurigo is more common in women.

Its seats of election are the posterior portion of the neck, the upper part of the thighs, the neighborhood of the genital organs and the intergluteal fold, the external surface of the legs, the popliteal and axillary spaces, the elbows and the posterior aspect of the forearms; but it may occupy any area, including the palmar and plantar regions. The focus is single, or there may be two, three or more foci.

At the onset, there is only intermittent itching, excited by occasional determining causes; next, it assumes the character of distinct, especially vesperal crises, lasting a few minutes, with furious scratching, followed by a voluptuous sensation of relaxation.

More or less rapidly, the lesions of prurigo develop and in these cases present a typical appearance, in the form of a patch of chronic lichen simplex of French writers, the lichen of Vidal of foreign authors. It is generally oval, averaging the size of the hand; three zones can be distinguished in it:

The external zone, 2 or 3 cm. wide, but imperfectly outlined, is brownish, quadrillated, barely thickened. In the middle zone appear lenticular and hemispherical prurigo papules, with an excoriated or shining surface grouped near the central zone. The latter is an infiltrated, hyperchromic or depigmented patch, lichenized to the highest degree, with a scaly or macerated epidermis according to the region and more or less distinctly outlined borders.

Frequently the patches are not complete; the zones which represent the successive stages of the change may be missing on one side, or altogether; the central disk may be replaced by more or less closely agminated papules.

The duration of a circumscribed prurigo is from several months to one or two years, sometimes still longer. Recurrences are common. Sometimes, one or several new patches appear when the old ones have ceased to itch, become flattened and smoothed out; but the normal color is not restored until long afterward.

The differential diagnosis must be made from lichen planus, lichen obtusus, the eczematides, the tubercular syphilides; it is usually easy, especially with the help of the anamnesis. In addi-

tion to the rather common nerve or less marked pigmentary disturbances, or leukomelanoderma, cases of circumscribed prurigo occur in association with true vitiligo.

Treatment of Pruritus and Prurigo.—Needless to say, before beginning the treatment of a pruritus or prurigo, the diagnosis of its form and probable cause must have been specified as closely as possible.

In the first place artificial pruritus, of parasitic origin or due to external causes, must be eliminated; these require a parasiticide medication or special hygienic precautions; next, secondary pruritus, in which not only the symptom but the primary disease and its complications require treatment.

In dealing with a primary pruritus or prurigo, its cause must be looked for among intoxications, hygienic errors, organic or functional anomalies; the urine and blood must be examined, etc.; it goes without saying that the treatment must take into consideration all the predisposing or determining factors which may have played a part.

In a general way, the alimentary regimen must be as simple as possible, in the form of a milk- or lacto-vegetarian diet, at any rate free from all foods or drinks of a stimulating character or of difficult digestion; it is especially important to insist upon careful chewing, cleansing the teeth and keeping them in good condition, counteracting constipation, etc. [The possibility that the underlying cause may be a sensitization to some article of food, often common like eggs or milk, must not be forgotten.]

The inherent nervous element of every case of pruritus must be treated by general as well as local measures and psychotherapy may have to be utilized.

Rest and a quiet mode of life are indispensable in overwrought or excited patients; the majority of sufferers from pruritus are benefited by a sojourn in the country, in the mountains or by the seaside. It has been observed that in the poorer classes, interrment in a hospital usually brings about rapid improvement, whereas their discharge is followed by a prompt recurrence. Spinal puncture, as tried by Thibierge and Ravaut, is sometimes followed by a sudden and permanent improvement in these cases. The modern practice is to recommend various sero-therapeutic procedures, injections of normal human serum, saline infusions, auto-hemotherapy, hypertonic sera with glucose or magnesium salts, etc., although the indications are not yet clearly established.

Sedative medicinal agents, bromides, valerian, antipyrin and its analogues, carbolic acid, salicylates, etc., are of little value. Extract of guaco is very unreliable. As a rule, tonics are called for; cod-liver oil and arsenic, persistently administered, are often

very useful in Hebra's prurigo, in the neurodermatitides and the prurigo vulgaris of youthful debilitated patients.

Among physical agents, baths are often not well tolerated, even with the addition of bran, starch, gelatin, lime blossoms, vinegar, etc. It is infinitely preferable to utilize general showers either lukewarm or barely warmer than the skin, applied with a very gentle spray almost without percussion ("dew-like douches"), for two, three or even four minutes or longer, repeated once daily or even twice daily at the start.

Electricity, in the form of static baths, may soothe some cases of pruritus; high-frequency currents are even more useful.

Watering-places suitable for patients with pruritus are, according to the desired end: Bourboule, Néris, Laxeuil, Bagnères-de Bigorre, Saint Gervais, Louëche, Ragatz, etc.; sometimes, diuretic or strong sulphur springs are indicated.

Local treatment is also of great importance. Very hot rather than lukewarm washes should be recommended, with one of the antipruritic lotions indicated further on (Therapeutic Notes, section 4). Then it is essential to protect the pruriginous surfaces from light and external irritants; this is accomplished by means of occlusive dressings or more conveniently through application of plasters, zinc gelatin or pastes, salves or varnishes, according to the extent of the pruriginous surfaces and the regions occupied by it.

In the plasters and pastes, various tars may be advantageously incorporated, especially tumenol or vegetable tar, or antipruritic remedies such as menthol, phenol, camphor, various acids, etc. Unguentum glycerinii with tartaric acid and pure cod-liver oil in inunctions or in salves or plasters, also possess a certain value.

In circumscribed pruritus or prurigo two or three radiotherapeutic sessions (at a dose of 3 to 4 H every fortnight) are often strikingly successful, but their effect is not always very lasting; in case of repeated relapses, the applications must by no means be inconsiderately repeated, for fear of radiodermatitis. High-frequency currents act in a less brilliant fashion, but do not involve the same disadvantages; scarifications, filiform douches and douches of superheated air may be valuable adjuvants.

The essential point is not to regard any of these therapeutic procedures as a panacea and as sufficient by itself, but to employ them separately or together, according to the case, devoting special care to the discovery and treatment of the individual conditions [the causative factor] in a given case.

[I would particularly recommend the following plan of treatment for the old patches of lichen simplex chronicus: The patches are vigorously rubbed with a 10 per cent. solution of caustic potash till the surface begins to be denuded of epithelium and shows many

oozing points. The burning sensation thereby produced is rather agreeable to the patient. The surface, washed with water and rapidly dried, is then painted with a solution of equal parts of ichthyol (or thigenol) and water and dusted thickly with an indifferent powder. The following day, treatment with the strong salicylic-acid-chrysarobin-tar ointment proposed by Dreuw is begun; an application is made night and morning for five days; then follows a period of four or five days during which the patient washes the affected area daily with soap and applies Lassar's paste. At the end of this period the discoloration produced by the Dreuw's ointment will have worn off and the patient is ready to begin the course again, starting once more with the caustic potash application, which should always be made by the physician himself.

For the temporary relief of itching in all forms of prurigo, raying with ultraviolet light is of decided value.]

CHAPTER XXV.

PARASITIC DERMATOSES.

Numerous parasites, animal or vegetable, may dwell on or in the human skin, giving rise to very polymorphous affections.

The *animal parasites*, which are sometimes divided into *epizoa* and *dermatozoa*, comprise insects, acari and worms.

The *vegetable parasites* or *dermatophytes*, with the exception of the schizomycetes or bacteria, belong for the most part to the class of mucedinia or filamentary fungi. Only the mucedinia which vegetate in the epidermis and its adnexa, the hairs and the nails, will be discussed in this chapter; the affections which they cause may be designated as epidermo-mycoses. As to the true dermato-mycoses, diseases due to dermatophytes growing in the corium, a special chapter will be devoted to their discussion (Chapter XXVIII).

DERMATOSES CAUSED BY INSECTS.

The parasitic insects of man live for the most part on the surface of the skin; they are *epizoa*.

Pediculosis and Phthiriasis.—Under these names are designated the cutaneous lesions produced by lice.

Lice are insects of the genus aptera and the family of pediculi. They have a pyriform head, provided with a sucking rostrum and mandibles which can seize the skin; a thorax bearing six feet terminating in a movable hook; and an abdomen not clearly separated from the thorax. The females, more numerous and somewhat larger than the males, lay a large number of eggs, with a chitinous envelope, which are designated as *nits*. When they hatch, the young resemble their parents and undergo no metamorphosis.

Three kinds of lice are parasitic on man:

Head-lice.—The head-louse, *pediculus capitis*, is about 2 mm. long, of a rather slender shape, a gray color marked with black spots on the border of the abdominal segments. It inhabits the scalp, especially in children of both sexes and in careless women and rarely the beard in men. In schools and asylums for pauper children, lice are endemic; in the seventeenth and eighteenth centuries, they flourished even at court in the heaped-up coiffures of the great ladies.

By their bites, lice cause severe itching, scratching and excoriations; but these symptoms may be altogether absent in some individuals. Without proper care, these lesions become infected and instead of simple crusted papules, impetigo, yellowish crusts adherent to the hair, folliculitis and abscesses of the scalp, pyodermatitis and adenitis at the nape of the neck and the lateral cervical regions, eczematization of the nape of the neck, the ears, the face, etc., may be seen.

In a social environment of absolute neglect, the head of some individuals may be found to be covered as with a cap formed by matted hairs studded with innumerable nits, teeming with lice, matted together by infected crusts with a nauseating odor; the underlying scalp is bathed in pus. These extreme cases are designated under the names of *trichoma* or *plica*.

The deep follicular inflammations and abscesses may leave a patchy cicatricial alopecia, which is incurable. The glandular suppurations of the neck have been known to lead to anemia, cachexia and generalized infections.

The lesions of pediculosis of the scalp begin and predominate in the occipital region; a pruritus or impetigo localized at this point is suggestive of lice and these should be looked for, irrespective of the age or social standing of the patient. If they are not readily discovered on parting the hair, at least nits may be found appearing as white or grayish oval grains stuck fast to the hairs in more or less considerable number.

Fig. 151.—Pediculus capitis—male. After Küchenmeister.

Treatment.—In boys or men, the hairs should be clipped close to the head; in young women, the hair can almost always be spared.

In those cases where the hair teems with lice, it is customary in the Saint Louis Hospital to begin the treatment with the application on the head during one night of a thick layer of vaselin covered with a bandage; the vaselin smothers and destroys the parasites.

When the lice are not so abundant, dressings with camphorated alcohol left in place for a few hours, or washing with bichloride in vinegar (1 : 500), or applications of powdered stavesacre or pyrethrum sometimes suffice to kill the lice and nits. To get rid of the latter, it is advantageous after untangling the hairs to employ a fine-toothed comb dipped in hot vinegar; the latter has the property of dissolving the cement which fastens the chitinous envelope of the nits to the hairs and of facilitating their removal. When there are many crusts, these should be softened with sprays or moist dressings, before removing them with soap and water;

followed by the application of a sulphur, naphthol or salicylic acid ointment or balsam of Peru.

Body-lice.—The body-louse or clothes-louse, *pediculus corporis, seu vestimenti*, is longer, measuring nearly 3 mm., of a yellowish white color and its abdomen has no black spots. It inhabits especially the clothing in contact with the skin, flannels, shirts and drawers; it is encountered among the poor, in tramps and in outcasts neglectful of all personal cleanliness. Unlike the head louse, it attacks adults and old people rather than children. Lice have abounded in the trenches during the war. The parasites or nits are found especially in the folds, along the seams, where they are accumulated in severe cases of pediculosis as a dense mass of yellowish beady granules, stuck to the threads of the garment. They are also found, however, on the body hairs, notably at the pubis, in 90 per cent. of the cases (H. Bulliard, 1917). The clothes-louse is proverbially prolific; according to Leuwenhoeck, two breeding females may in the course of two months produce 18,000 young lice.

Fig. 152. — Pediculus corporis—female. After Küchenmeister.

The bite of the body-louse gives rise to a highly pruritic urticarial papule. The itching and scratching are especially severe in the evening and at night. A sort of habituation apparently becomes established in inveterately lousy subjects, there is no longer an eruption and the itching is not conscious, although the scratching persists instinctively, as shown by the linear excoriations following the scratch of the nails. The shoulders and the upper portion of the back represent the site of election of this pediculosis, next the abdomen, the loins and the anterior aspect of the thighs. The face, the scalp and the feet and hands are exempt. The excoriations may become the starting-point of pyodermatitides.

[Body-lice have been shown to be carriers of typhus and possibly also of "trench-fever."]

A long-standing pediculosis leads to a rather peculiar modification of the skin, characterized by thickening, a dry and scaly epidermis and especially a dark pigmentation; from this background stand out the white cicatrices, recent excoriations and the innumerable crusts. This *melanoderma* of pediculosis (p. 329) is most marked on the shoulders and the back, but may become generalized. The pigmentation may be observed even in the mouth, a proof that scratching does not sufficiently explain its genesis.

A differential diagnosis in these cases must be made from Addison's disease, in which pigmentation of the mouth is usually present. But the topographical distribution of the Addisonian melanoderma is different and itching and evidences of scratching are absent.

Asthenia and cachexia possess less diagnostic value, being, likewise encountered in the chronic pediculosis of poverty-stricken subjects, which here assumes the appearance of a general disease, known as Vagabonds' disease.

The differential diagnosis from the diathetic forms of pruritus must take into consideration the different localization of the itching, but is based especially upon the presence or absence of the parasites and their nits.

The *treatment* consists in disinfection of the clothing in a steam sterilizer and in the necessary personal cleanliness.

When no steam sterilizer is available, the garments may be pressed out with a hot iron or exposed to fumigations with cinnabar; these measures, combined with the persistent employment of insecticide powders, the wearing of clean linen and repeated washing with soap and water, followed by parasiticide inunctions (see Therapeutic Notes, section 10) may be sufficient.

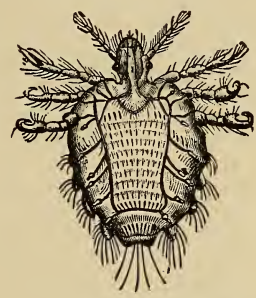


Fig. 153. — Pediculus pubis. After Schmarda.

Pediculus Pubis. — *Phthirius inguinalis*, popularly known as the crab-louse, is nearly as broad as it is long, vaguely resembling a crab; it lives on the hairy regions of the pubis and vicinity, where it clings to two neighboring hairs by means of the curved hooks of its feet. In hairy men, it may invade the thighs, the entire trunk and the beard; in both sexes it infests the axillæ; in children and young women it may even lodge on the eyelids, causing a blepharitis phthiriasica.

Crab-lice are usually acquired through sexual intercourse, but also through indirect contagion, in water-closets, or infested bedding in hotels. It has often been noted that this parasite is less common in the very lowest class where personal cleanliness is absent, than in the middle and even in the wealthy class.

Pediculus pubis gives rise to a very variable amount of itching in different individuals and to scratching with its usual sequelæ. This etiology must be kept in mind in considering an eczema or pyodermatitis of the regions mentioned. Together with the crab-lice, nits will be discovered sticking to the hairs near their base.

A peculiar consequence of the bite of phthirius is the appearance

of *blue spots* or *shaded spots* [taches bleues, taches ombrées, *maculæ ceruleæ*], seen especially on the abdomen, the flanks and the thighs. Formerly regarded as symptomatic of undetermined or even of typhoid fever, these spots have been traced to their true cause by Falot, Mourson and the experiments of Duguet [though their pathogenesis is still unknown].

Treatment.—The classical treatment with inunctions of blue ointment—which should be rejected on account of the mercurial eruptions so frequently [and stomatitis occasionally] caused by this salve—is preferably replaced by inunctions with yellow precipitate ointment of 5 or 10 per cent., or a white precipitate or naphthol salve or balsam of Peru. Washing with bichloride-alcohol (1 : 500) is also effective. [A salve containing three parts of ammoniated mercury ointment and one part of blue ointment is safe and effective.] In phthiriasis of the eyelids, Jullien recommended the removal of the parasites one by one by means of a forceps.

Other Sucking Insects. — **Fleas.** — These are of various kinds, each attacking a different animal. The human flea, *pulex irritans*, lays its eggs in the dust of floors, etc. Its bite produces a characteristic lesion; a hemorrhagic point surrounded by a lenticular zone of erythema; this disappears in a few hours, whereas the central ecchymosis persists several days. In children and in persons with an irritable sensitive skin, an urticarial wheal forms at the onset; [and in some cases, the wheal on subsiding leaves behind a papule which itches periodically for several days]. Some individuals are not attacked by fleas. In cases of extreme abundance of fleas, in rag-pickers for example, the entire skin may be dotted with minute red spots resembling purpura.

Fleas may act as carriers of the germs of severe infectious diseases; there is reason to believe that these insects convey Bubonic plague from rats to man.

Pulex Penetrans or Jigger.—This variety, *Rynchoprion penetrans* or *Dermatophilus penetrans*, occurs in tropical America and in Africa; the female burrows into the cutis, especially on the toes and soles of the feet, where it grows to the size of a pea, causing a furunculoid abscess. The loss of one or several toes, gangrene and tetanus, may be the consequence of this "dermatophilia."

Bed-bugs.—The bed-bug, *cimex lectularius*, lives and multiplies in wooden bedsteads, in cracks of wainscoting and in upholstery. At night these animals sally forth from their hiding place and bite the sleeper, producing red urticarial elevations on the skin, sometimes with extensive edematous swelling and a painful burning sensation. They are suspected of being possible carriers of Oriental boil, Bubonic plague, the trypanosomiases and Koch's bacillus.

Mosquitoes.—This name is applied to an entire series of species of *culex, simulia, stegomya, anopheles,* etc., distributed in all countries, especially hot countries. The female sting the parts accessible to them, especially at night, producing a rather persistent urticarial swelling, particularly after scratching.

Great interest is attached to these insects since it is known that their various kinds serve as intermediary hosts and transmitters for the parasites of malaria (anopheles), of yellow fever (stegomya), of filariasis and perhaps also of pellagra (simulides) and of leprosy. [The simulium theory of pellagra is no longer entertained.] Through the systematic destruction of mosquitoes immense tracts of land have been redeemed.

The *treatment* of the bites of fleas, bed-bugs and mosquitoes consists in applications of alcoholic solutions of menthol or carbolized vinegar, dilute ammonia, collosol, naphthalan, etc., followed by bland powders. General prophylaxis, namely the destruction of the insects, often presents serious difficulties. Protection against mosquito bites is secured through the employment of mosquito-netting [or by rubbing the exposed parts of the skin with oil of citronella]; in a closed room, pyrethrum may be burned.

DERMATOSES CAUSED BY ACARI (MITES).

The parasitic acari of men are in part true dermatozoa, living in the horny epidermis, such as acari or sarcoptes, or in the pilosebaceous follicles, such as demodex; and in part epizoa, such as ticks, etc.

Scabies.—Scabies is a contagious parasitic dermatosis, caused by an acarus, the *Sarcoptes scabiei* (Latreille), variety *hominis* (Mégnin). It is extremely pruritic and is characterized essentially by a special dermatological lesion, the burrow; and by polymorphous accessory eruptions, having a regional and symmetrical distribution.

The ancients, confusing scabies, psora or the itch, with the prurigos, interpreted it as a diathetic disease, subject to metastases, which was treated by venesection and blood purifiers. It was in vain that the parasite was pointed out by Mouffet (1634), C. Bonomo (1687), Wichmann (1786). The truth was not established until Renucci (1834), a student from Corsica, demonstrated in public in the clinics of Alibert, the sarcoptes which his countrywomen had taught him to know and extract (Fig. 154).

Symptoms.—The itch is observed in all social strata and at any age; it is more common, however, among prostitutes and paupers living in crowded quarters.

The symptoms do not appear until after a latent period, averaging ten days.

The eruption is localized or predominates in certain areas of predilection, which should be systematically explored in all patients

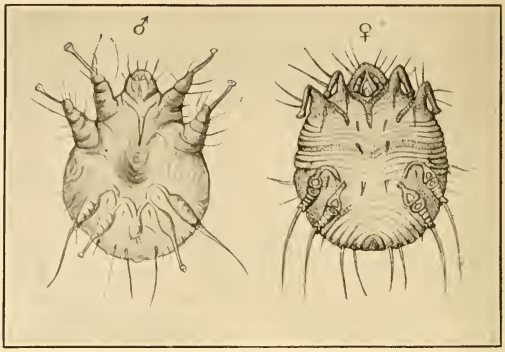


Fig. 154.—Sarcoptes scabiei, male and female. Reduced from Furstenberg, after Murray.

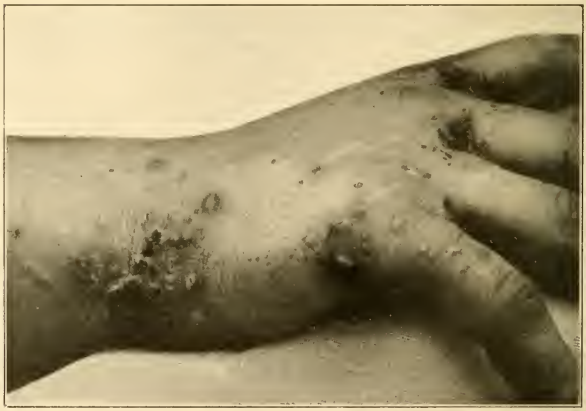


Fig. 155.—Scabies.

complaining of nocturnal itching. These are: the hands (Fig. 155), the interdigital spaces, the lateral aspects of the fingers, the wrists, more particularly their ulnar side, the elbows, the anterior wall of

the axilla, the ankles and the heels; in men, the sheath of the penis and the glans; in women, the breasts; in children, the buttocks. But the lesions may also occupy any other region, excepting the head which always escapes and the neck and back which are usually free. This topography in itself is characteristic.

The itch, however, has a pathognomonic feature, the burrows. These are narrow grayish tracts, as if traced with the point of a needle, taking a curved or sinuous course which does not correspond to the folds of the epidermis. They have a length of 2 to 3 mm. or more; Dubreuilh observed a burrow 4 cm. long on the foot. These burrows represent the passages dug by the parasite in the horny ayer of the epidermis. They are often dotted with black points which indicate the exit orifices of the newly hatched larvæ. Their

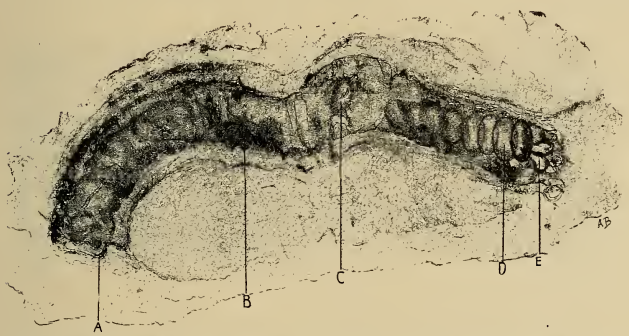


FIG. 156.—Scabies burrow. The shred of horny epidermis containing this burrow was ablated with a razor; it is shown from its lower or deep aspect. *A*, entrance orifice; *B*, black matter, excreta; *C*, opening; *D*, ova; *E*, acarus. × 50.

general color, which is darker in laborers and uncleanly persons, is partly referable to the excreta left by the sarcoptes and also to deposits of dust and dirt. White burrows may be found in the better classes.

One of the extremities of the burrow, its head, is marked by a small nacreous elevation, the *acarus eminence* of Bazin; this is a tiny deep vesicle which forms in the vicinity of the sarcoptes. The animal may be extracted by tearing off the roof of the burrow with a needle and catching it in the cul-de-sac; or the entire burrow may be excised by means of fine curved scissors or a razor (Fig. 156). The burrows are most easily discovered on the fingers, the wrists, the elbows, the penis, the heels, in general where there is a thick epidermis,

The other lesions of scabies are accessory or secondary eruptions, of variable characters and abundance. At the onset, these consist of spots of erythema or urticaria; next, of punctate or linear excoriations produced by scratching, crusted miliary papules, vesicles of dysidrotiform eczema, or ordinary eczema scattered or in patches (scabietic eczema); and finally, of various pyodermatitides —impetigo, folliculitis and ecthyma (scabietic ecthyma), suppurating or dried into crusts which are scattered by the scratching and may even be transported to the face. These lesions may become complicated by lymphangitis, adenitis and cellulitis. Eczema of the nipples and areolæ in women (Fig. 157) is always due either to scabies or to pregnancy. The polymorphism of the scabies eruptions is a characteristic feature, but their topographical distribution is even more so.

Fig. 157.—Scabies eczema of the breasts.

The itching of scabies is usually almost intolerable. Its principal attribute is its nocturnal character; it is especially pronounced at the moment of retiring, but lasts until morning, often causing complete insomnia. This time is the period of activity for the acari. The itching is more or less localized or general. In some individuals, scratching is unconscious; they scratch and flay themselves without being aware of it. Pruritus may be absent although this is very rare. [A moderate degree of eosinophilia is commonly present in the blood of scabietics.]

Etiology.—The fertilized female of *Sarcoptes scabiei* is alone responsible for digging the burrows and causing the symptoms of scabies. It has the shape of a flattened oval and measures one-third or one-fourth of a millimeter; it is therefore visible to the naked eye as an opaque whitish point. The anterior two pairs of its eight feet are furnished with cupping pads, the posterior two pairs with long bristles. The males, which do not inhabit the burrows, are much rarer and more difficult to discover; they are only half the size of

the females and their fourth pair of feet also is furnished with cupping pads.

The ova, which the female drops behind her in the burrow, measure 0.16 mm. x 0.10 mm. and are more developed in proportion to their nearness to the entrance orifice. They hatch in four to eight days and the young leave the burrows through the openings to settle elsewhere.

The sole cause of scabies is the transmission of the parasite. There exist neither individual immunity nor predisposition. In view of the habits of the sarcoptes, the contagion occurs almost exclusively at night. It is necessary for an impregnated female to pass from an infected to a healthy subject. This transmission is practically inevitable in persons who share the same bed, hence family epidemics and the frequent venereal origin of scabies. Contagion is possible through unchanged bed-sheets in hotels, through clothing, through sheets in sleeping-cars; it may very exceptionally result from contact in daytime; it is very doubtful if it can occur through tools, books, etc.

[Scabies is one of the commoner skin diseases. In the statistics of American Dermatological Association it averaged about 5 per cent. of all cases during a period of thirty years. In the first decade of this century, however, there was a great increase in the incidence of the disease and in 1908 it constituted 10 per cent. of all skin diseases seen—a veritable epidemic. In general, scabies is somewhat more frequent in European countries than in America.]

Prognosis.—Scabies never heals spontaneously. In some nervous individuals it may end in marasmus, on account of the pruritus and insomnia. Albuminuria is said to be not very rare in patients suffering from the itch; it is attributed either to the toxins of the sarcoptes or to nephritis caused by the microbes of suppuration.

In certain villages of Norway, Brittany, Italy, scabies was endemic for a time; it persisted throughout life, usually without causing severe symptoms.

In the course of febrile diseases, the symptoms of scabies as a rule entirely disappear, recurring as soon as convalescence sets in.

Diagnosis.—The topography and polymorphism of the eruptions, the nocturnal pruritus and the contagiousness, make the diagnosis probable; the demonstration of burrows or of acari or their ova makes it positive.

The essential point is to keep scabies in mind, whatever the environment of the patient; not allowing one's self to be deceived by the eruptions of strophulus, prurigo, dysidrosis, pediculosis, eczema or pyodermatitis.

A real difficulty results from the obsession known as *acarophobia*, or fear of the itch; this is observed in persons who have heard of it

and especially in those who have had the disease and been treated for it. When pruritus and eczema persist, after treatment by the method called "la frotte" to be described presently, baths and soothing creams should be recommended instead of hastening to resume the treatment, unless new burrows are discovered.

Treatment.—In order to cure the itch, it suffices to destroy the parasite and its ova; no internal treatment of any kind is required.

The classical "frotte" of the Saint Louis Hospital constitutes the most rapid treatment.

It begins with rubbing the whole body vigorously with soft soap for twenty to thirty minutes and continuing the friction in a warm bath for another hour, in order to open all the burrows; during this time the patient's clothes are disinfected in a steam-sterilizer. Next, the body is thoroughly rubbed with the Helmerich-Hardy ointment, which is left on until the next day, when a full bath is administered.

Less irritative sulphur ointments may be utilized, according to the formulas of Bourguignon or Fournier (for examples see Therapeutic Notes, §10).

The "frotte" treatment must not be applied to young children, pregnant women, patients with a very delicate skin or those suffering from extensive pyodermatitis. In such cases other substances may be utilized, such as balsam of Peru, styrax, naphthol, etc., which have been tried and approved but which must be employed in moderate strength for eight to ten days in succession, at night, preferably, after washing with soap; disinfection of the clothing is not indispensable.

Care must be taken always to examine the bedfellow and children of the patient, as well as to treat on the same day all who have been discovered to be infected.

Animal Scabies.—*Scabies Norwegica* or *crustosa*, described by Danielssen and Boeck in lepers, also occurring in Germany and various other countries, gives rise to thick and prominent incrustations which may occupy the entire body, including the back and the face. In all probability this condition represents not simply an inveterate common itch, but a special parasitical variety, as was maintained by Mégnin, the sarcoptes of wolves.

Other animal itches, caused by sarcoptes other than the human variety, are very rarely transmitted to man. The absence of burrows has almost always been expressly noted. The eruption is miliary or polymorphous, diffuse and pruriginous. These abnormal itches may be derived from cats, birds, dogs, sheep, goats, camels, pigs, etc. They are usually very readily curable or even subside spontaneously.

The most noteworthy and serious form is *equine scabies*, of which

I observed an example almost identical with the case published by Besnier and Mégnin in 1892. The appearance was that of a pityriasis rubra, completely generalized; the sarcoptes were demonstrable by thousands in the scales and crusts; there were no burrows.

Other Parasitic Acari.—Demodex Folliculorum.—This is a worm-like acarus, measuring from 0.3 mm. to 0.4 mm., whose cephalothorax is provided with a mouth and four pairs of rudimentary feet; the abdomen resembles the finger of a glove and shows fine transverse striations.

This parasite inhabits the sebaceous follicles, especially the mouths of the large sebaceous glands of the face, with its head directed inward; a large number, ten or twelve, may be found in the same follicle.

It has the reputation of being non-pathogenic, causes no inflammation and certainly plays no part in comedo and acne vulgaris. It was held responsible, however, by Dubreuilh, in a case of localized pigmentation; personally, I have seen it so abundantly present in the horny prominences of a lichen spinulosus that I was tempted to attribute an irritative influence to it in this case.

Certain observations of Borrel would seem to suggest that the demodex, or analogous acari, might intervene in the etiology of epitheliomas of the face, as irritants or as carriers of a hypothetical contagium, as well as in the etiology of leprosy. A disease of dogs, known as follicular mange, not transferable to man, is due to a variety of demodex.

Leptus Autumnalis (rouget or aoûtat) is the larva of a trombidium holosericum which lives on vegetables, notably on beans, wild grapes and many varieties of grasses. Certain regions have for a long time been infested with it; at present it is spreading in numerous localities in the vicinity of Paris. These mites flourish in the summertime and attack those visiting fields or gardens; attaching themselves to the legs, the thighs, the waist and the axillæ, especially where a band such as a garter or belt, etc., retains them; but they are found even on the ears and the face. They cause a frightfully itching urticarial papule which the patients scratch off with their nails. Careful inspection, preferably with a lens, reveals the parasite in the form of a blood-red dot, measuring from $\frac{1}{4}$ to $\frac{1}{2}$ mm. in the follicular mouth of a hair; often several are seen together. Under the microscope, they resemble cheese-mites, but of a red color.

Infection may be guarded against by rubbing with tincture of benzoin or oil of cade. The treatment of the very distressing eruption caused by the parasite consists in washing with benzene or applications of Peru balsam or tincture of iodin.

Ixodes (Ticks).—The most common (in Europe) is *ixodes ricinus*, the wood-tick, a large brownish acarus which in the fasting state measures from 3 to 4 mm.; its body becomes globular and considerably swollen when the animal is gorged with blood. It attacks dogs, cattle and large game, but rarely human beings.

Argas.—Argas reflexus marginatus, closely related to the ixodes, is of similar size and has the appearance of a small gray shield. It is a pigeon-parasite and infests dove-cots. When it accidentally bites man, a very painful extensive phlegmonous edema has been known to follow, with vesiculation or generalized urticaria, distress, tachycardia and digestive disturbances; these symptoms are undoubtedly due to inoculation with septic germs. Other species of *argas* and *ornithodorus* may transmit to man various pathogenic agents, notably the spirochete of "tick-fever," etc.

Pediculoides Ventricosus.—In laborers engaged in unloading exotic cereals from various countries and on the boats which carry these cargoes, one may observe, sometimes in epidemic form, an extremely pruritic erythemato-papular eruption caused by the acarus which bears this name. It measures from 120 to 200 μ. in length by 70 to 80 μ. in width. Washing with dilute vinegar suffices to cure these patients. [The parasite is conveyed in wheat straw and considerable endemics of an urticarial disease have occurred in consequence of the use of the infested straw for making cheap mattresses.]

DERMATOSES CAUSED BY WORMS AND LARVÆ.

Among the numerous parasitic worms of man, only one really lodges in the skin, namely filaria medinensis.

Filaria Medinensis or Dracunculus.—This is a nematode worm occurring in many tropical countries, notably in Western Africa (*guinea-worm*).

The adult female is filiform and from 60 to 80 cm. long; it remains rolled up in the frequently single abscesses which its presence produces, usually on the feet or legs. The filaria is ingested by drinking stagnant water in which the embryos have been deposited and have penetrated into a small crustacean, a cyclops (Manson) [which serves as an intermediate host]. It is not until a year later that a fecundated female filaria emerges at the skin.

In place of the classical *treatment*, namely extraction of the worm by pulling and rolling it up on a small stick, in which rupture is a common and dangerous occurrence, it has been recommended to inject a solution of bichloride, 1 to 1000, in the vicinity of the worm, which dies and is then more easily extracted.

Filarial elephantiasis (Chapter XVIII) cannot be interpreted simply as a parasitic dermatosis.

Various eruptions have been reported as due to other kinds of filaria. *Craw-craw* is a pruritic papulo-vesicular, later ulcerative dermatosis of the feet and legs, observed in Western and Central Africa. It is imperfectly known and has been referred to microfilaria (O'Neil), larvæ (Nielly), etc.

Pani-ghao, or *ground-itch* of Assam and the United States, is a papulo-vesicular and pruritic dermatitis due to the larvæ of ankylostoma duodenale [hook-worm] having penetrated into the skin of the feet or legs.

Cysticercus Cellulosæ.—This parasite is sometimes present in the subcutaneous tissue of man as well as pigs and is apparently more frequent in Germany than in France [and very rare in America]. The cysticercus cellulosæ is the vesicular form of *tenia solium*. The parasites appear as small and hard, round nodules, the size of a large cherry pit, present in more or less considerable number in the hypoderm or more deeply, causing neither pain nor inflammatory reaction. They contain a clear fluid and a scolex provided with its hooks and suckers. The tapeworm whose ova have caused the infection not infrequently inhabits the intestine of the patient himself or that of a person in his environment.

The treatment requires the extirpation, or better, electrolysis of each nodule with the positive pole.

Larva Migrans.—Under this name is designated the parasite of a very peculiar affection named creeping disease, pointed out by H. G. Lee, in 1874 and 1884. It is characterized by a red line, 1 to 3 mm. wide, irregularly winding and undulating on the skin, sometimes forming knots and loops; its extremity advances from 1 to 10 cm. daily. This dermatosis is not uncommon in Russia. It is difficult to seize the parasite; it is a very motile, black-headed, 1 mm. long larva of a *gastrophilus* or horse-fly. The affection may be cured by tincture of iodin or bichloride of mercury [by injections in front and around the head of the advancing line.]

Various *cutaneous myases* due to the larvæ of flies (oestridæ and muscidæ) are related affections.

EPIDERMOMYCOSES.

The epidermomycoses are caused by mucedineæ and are purely local affections, without reaction upon the general health.

Among the thread fungi which are entitled to the name of *epidermophytes*, some affect preferably the scalp (*tinea* XX) or the beard (*sycosis*, XIX) and the nails (*onychomycoses* XXI).

The same species may also vegetate upon the hairless skin, while

others affect it exclusively; in this way the *erythemato-squamous* (V) and *eczematiform mycoses* (IV) originate.

Favus.—Under this name is designated an epidermomycosis due to the *achorion Schönleinii*.

Favus, formerly confused like the tineas with porrigo and impetigo, was separated by Biett and the brothers Mahon. Its parasite was discovered by Schönlein of Zürich, in 1839, well described by Gruby and christened by Remak.

Favus is essentially a rural disease, is not limited to children and produces crusts and cicatrices; a spontaneous cure is exceptional. These features differentiate it from the other epidermomycoses.

The characteristic eruptive lesion of favus is the *scutulum*, a saucer-shaped crust 2 to 4 mm. in diameter, thick in proportion, of a sulphur yellow color, usually pierced by a thick or downy hair.

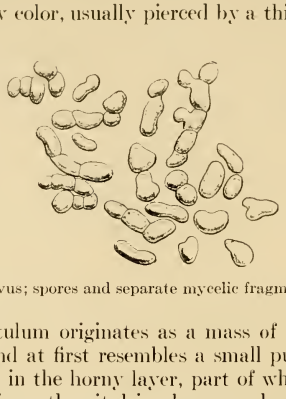


Fig. 158.—Favus; spores and separate mycelic fragments. × 900.

The cup or scutulum originates as a mass of white matter in a follicular funnel and at first resembles a small pustule. This mass grows and extends in the horny layer, part of which covers it for a certain length of time; then it dries, becomes depressed, yellow and friable. In three weeks the cup attains a diameter of 3 mm., and may grow still larger. More frequently, neighboring cups of different sizes become agglomerated into an irregular roughened mass which has been compared to a honeycomb (favus) and has a mousy odor.

The material of a favus-cup is easily broken up into a grayish dust. Examined under the microscope, in a drop of 40 per cent. caustic potash or of formic acid, the cup appears composed of spores in great numbers and short tubes of mycelium (Fig. 158). The mycelial segments, from 4 to 15 μ. long, from 3 to 7 μ. wide, are of irregular form, present short lateral branches and often terminate in a row or tuft of cubical members or spores. These spores are rounded, oval or irregular, and of varying diameter; they consist like the tubules of a granular protoplasm and a not easily differentiated membrane.

The achorion is characterized by its abundance as well as by the irregularity and clumsiness of its forms.

In a cross-section of a favus cup (Fig. 159) which is made up entirely of parasites, one finds at the base, slender imperfectly partitioned threads; in the middle layer, thick sporulated threads, at the surface, short members and spores; the whole may be covered with lamellæ of horny epidermis.

Malassez observed in a case of favus that the mycelial tubes penetrated under the cup as far as the connective tissue of the cutis, without exciting a special inflammatory reaction. His specimens, which I was enabled to study, are convincing and have been endorsed by all histologists who have seen them; but such a penetration is exceptional and has never again been demonstrated by myself or by others. It certainly does not explain the inflammatory reaction caused by favus and the cicatrices which it leaves behind.

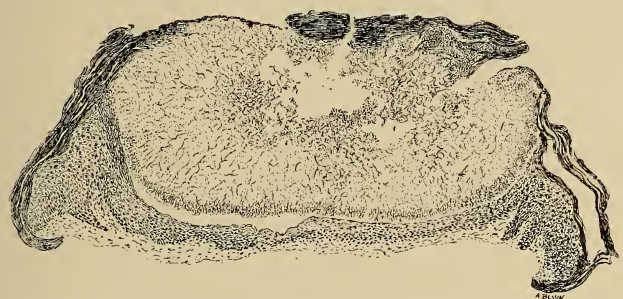


FIG. 159.—Favus; cross-section through a cup imbedded in the epidermis. × 60.

I have personally described with J. Hallé (1910), under the name of *granuloma favosum*, intradermic tuberculoid nodules surrounded by a ring of giant cells, found on the scalp in a case of favus. These are very rare and are probably due, like the trichophytic granulomas of Majocchi, to the destruction of hair follicles.

In a scale of epidermic favus without cups there are found not only a very irregularly partitioned mycelium but also collections of spores which are not seen in herpes circinatus trichophyticus.

The dull and discolored hairs, in favus, are not brittle like trichophytic hairs, because the parasite is much less abundant in the favus hairs. One finds in them a variable number of greatly twisted tubules, either thick and sporulated, or slender and more rectilinear, composed of segments from 12 to 15 μ in length, dichotomized at an acute angle and generally surrounded with air-bubbles, which at once indicate their presence.

In favus of the *nails*, the ungual cells are dissociated by irregular, slender or very bulky filaments or by spores; this appearance is not characteristic.

Cultures of a particle of a cup or of the root of a favus hair, on peptonized agar, yield in three or four days, at a temperature of 90° F., a star of white radiating filaments; by the end of three weeks this has become an irregular patch, 2 to 4 cm. in diameter, with an irregular undulating surface, of a yellowish gray color, smooth or covered with a scanty whitish down; its periphery sends out branching and nodular radiations.

The achorion Schönleinii belongs botanically to the genus *oöspora;* it is responsible for practically all cases of human favus. Due to the remarkable investigations of Sabrazès, Bodin and others, we know at the present day several other species of achorion in animals —mice, cats, dogs and poultry (achorion Quinckeanum, gallinum, gypsœum, oöspora canina); but these have been very rarely met with in men, in trichophytoid eruptions, sometimes with minute cups, but never on the hairy scalp.

Etiology.—Favus is contagious, although to a less degree than trichophytosis and is rarely epidemic. It is hardly ever contracted otherwise than in the course of childhood, from five to fourteen years, at school or at home; it frequently happens that of several children who live together only one or two are attacked. As favus has practically no tendency to a spontaneous cure, it may persist throughout life and become perpetuated in certain families and localities.

Transmission from one human being to another is the rule and may be direct or through the medium of clothes, head-coverings or toilet articles. Favus of animal origin, as stated above, is extremely rare.

Favoring conditions are dirt, poverty and promiscuity. In Paris, cases of favus are almost without exception imported from the country or from abroad. The disease tends to diminish in France; it is still most frequently met in Lyons, in the northwest and in the south. Favus is rare in England and America, but common in Poland, Italy, Holland and Algeria.

Clinical Forms.—Tinea favosa (XX) and onychomycosis favosa (XXI) have already been described. I have also pointed out the characteristics of the alopecia and cicatrices due to favus (XVII).

Favus of the glabrous skin is not very rare, but is found especially in persons whose scalp has first been infected, so that it is generally the result of auto-inoculations. The face, the shoulders, the abdomen, the buttocks and the external aspect of the limbs may be attacked. No positive case of favus of the mucous membranes is known; the observation of Kaposi and Kundrat, concerning a

favus patient who died with gastro-intestinal ulcerations, is open to question.

There are two varieties of favus of the hairless regions (Fig. 160); the *circinate epidermic* variety (favus herpeticus) closely resembles circinate trichophytic herpes, although it assumes a less regularly circular form; the epidermis of the red and scaly spots contains an abundance of short mycelial threads, spores and irregular tubules; the cupped variety develops as such from the start, or more often originates from the preceding form with the appearance of follicular yellow disks which grow at a rapid rate.

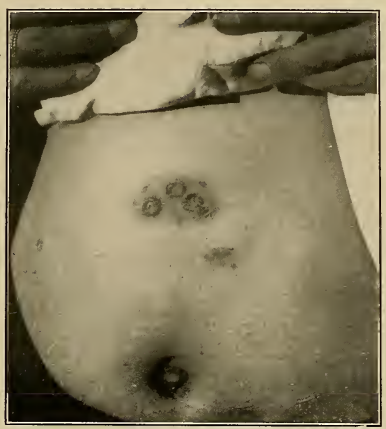


Fig. 160.—Favus of the hairless skin; the epigastrium of this young girl presents two spots, one of which bears four cups, the other being of the variety known as favus herpeticus.

Treatment.—Favus of the body is much more easily curable than favus of the hairy regions. It suffices to soften the cups with moist dressings, to remove them by scraping with the curette and to make two or three applications of tincture of iodin. As a rule, there remain no cicatrices, but only simple macules. Recurrences must be guarded against by carefully watching the patient. [The treatment of favus of the scalp has been discussed in chapter XX.]

Trichophytosis.—In a series of remarkable contributions, published from 1841 to 1845, Gruby established the parasitic character of the tineas. Credit is due to Bazin for the universal recognition of this fact. During fifty years one of these parasites, named the *trichophyton tonsurans*, by Malmsten, was assumed to be the sole

cause of tinea tonsurans of children, sycosis parasitaria of men, herpes circinatus trichophyticus and onychomycosis.

This view was altogether changed by the admirable investigations of Sabouraud, who confirmed the descriptions of Gruby, but applied bacteriological methods to the study of the tineas. He recognized the fact — confirmed after him by Mibelli, Bodin, C. Fox, M. Morris, etc. — that the pathogenic trichophytons of men and of animals do not belong to one and the same species, but constitute a family of numerous genera, varying in their morphology, their cultural features, their habitat and the clinical lesions they give rise to.

Fig. 161.—Mycelium of trichophyton, in a scale of herpes trichophyticus circinatus. Stained with Sahli's blue. × 270. For the inset, × 540.

Moreover, one of the parasites described by Gruby in porrigo decalvans is not botanically a trichophyton; it is called *microsporon* and the tinea produced by it bears the name of *tinea tonsurans with small spores* [microsporia] of Gruby-Sabouraud. It will be discussed further on.

Trichophytons. — The true trichophytons belong to the genus botrytis, family mucedineæ.

In their epidermic products, the trichophytons assume the shape of mycelium threads and spores. The mycelium is made up of elongated, slightly undulating, regular, colorless and transparent,

partitioned and branching tubules (Fig. 161); the partitions are usually very close together. In each compartment a rounded, oval or cubical *spore* is formed; these spores often remain in rows or strands.

For the microscopic examination of scales, hairs or nail clippings, it suffices to place these on a slide, add a drop of 40 per cent. potash solution, cover with a slip and heat gently over a flame to a temperature near the boiling-point. Formic acid also clears up the scales after a little time without heat. Amplification from 200 to 300 diameters is the most convenient. In order to obtain stained and lasting specimens, the epidermic particles must be rendered free from fat with alcohol and ether, stained with Sahli's blue and mounted in Canada balsam.

Trichophyton *cultures* are obtained without difficulty on agar media containing 1 gram of peptone and 4 grams of glycerin or sugar to 100 grams of water. Barely visible particles of hairs or scales should be planted aseptically and the test-tubes left without rubber caps at laboratory temperature. Very often the cultures are pure from the start, without its being necessary to begin by first washing the material in alcohol or silver nitrate, as recommended by some authors. After the third day, secondary cultures can be made, preferably on the *test medium of Sabouraud*, which gives the most distinctive physiognomy to the various species and is composed as follows: Granulated peptone (Chassaing), 10 parts; crude maltose (Chanut), 40; agar-agar, 18; water, 1000 parts. A temperature from 75° to 95° F. is the most favorable.

Besides the mycelium, the following organs of fructification of the trichophytons are found in these cultures: Spore-bearing hyphens usually in terminal clusters, exceptionally endoconidia and also, according to the species, more or less numerous multilocular spindles.

The number of known species of pathogenic trichophytons is about thirty.

It has been shown by the investigations of Plato (1902), followed by those of Truffi, that a *trichophytine* can be extracted from the cultures of several trichophytons, which when injected into a patient having trichophytosis with deep nodes will give rise to a cutaneous and a general reaction not occurring in healthy persons. An extract of achorion culture, or *favine*, seems to act even more constantly. These injections or applications of salves containing the toxins, might serve for the treatment of the deep trichophytoses. Bruno-Bloch and Massini have confirmed these observations and have, moreover, succeeded in immunizing animals against the tineas in general, by a preliminary inoculation with trichophyton gypseum or an animal achorion. Investigations in this direction now under

way promise important results from the standpoint of diagnosis, prophylaxis and treatment.

Clinical Forms.—The trichophytons give rise to various cutaneous affections, according to their localization on the scalp, the hairless skin, the beard or the nails.

Tinea trichophytica has been described elsewhere (XX). It may be due to several species. According to Sabouraud, it may be stated that at the present writing, of 100 cases of this affection in Paris, 50 are due to the *trichophyton crateriforme*, 30 to the *trichophyton acuminatum;* and the 20 remaining cases are due to various species among which the *trichophyton violaceum* predominates. In other countries, these proportions would undoubtedly be different.

The *granuloma trichophyticum* of Majocchi (1883-1906) is a rare form characterized by intradermic nodules, sometimes agminated or in strands, which develop in the cutis of the alopecic and squamous spots; they consist of a mass of inflammatory cells and a ring of giant cells surrounding fragments of trichophytic hairs. Pini and Lutati have observed a few similar cases.

Sycosis trichophytica of the beard, *kerion* Celsi and *folliculitis agminata* which have already been described in this book (XIX), are follicular, inflammatory and even suppurative forms of trichophytoses of animal origin. Their most common germ is the *trochophyton gypseum* of horses; sometimes it is a bird trichophyton with rose-colored cultures. These parasites are *ectothrix;* they possess pyogenic properties. Adults are more susceptible to the contagion than children; it results either from direct contact with the trichophytic animal—horse, dog, cow, birds, etc., whose lesions may not always be very evident—or indirectly from the handling of contaminated articles, brushes, coverings, etc. Transmission may then take place from man to man, by intermediation of the barber, for instance. The sequence of cases is often difficult to establish.

Jadassohn has pointed out in children suffering from kerion, the frequent co-existence of not very persistent folliculitides of the trunk, supposed to be of toxemic character and designated as *disseminated lichenoid trichophytosis.* The above statements on the etiology of sycosis are likewise applicable to that of *onychomycosis trichophytica.*

Cutaneous trichophytosis *of the hairless skin* was formerly known under the name of *herpes circinatus* which also comprised *cutaneous microsporia* and *epidermophytosis.*

Cutaneous trichophytosis is observed in:

1. Children suffering from tinea and persons in their environment; these cases are usually due to one of the ordinary endothrix trichophytons of the tineas. It is not uncommon, as was first pointed out by Besnier, that the epidermic manifestation which is very evident leads to the discovery of a previously undetected tinea tonsurans.

2. Persons of all ages, free from tinea, whose occupation or inclination puts them in frequent contact with animals. In these cases, the parasite is presumably an ectothrix of animal origin and does not invade the scalp.

Any region of the body may be involved; the seat of election of cutaneous trichophytosis, however, is the exposed parts, such as the face, neck, hands and forearms. Its palmar and plantar localization, of special appearance, has been mentioned under the heading of the keratodermas (p. 216).

Trichophytosis of the hairless parts manifests itself in the form of erythemato-squamous spots, noteworthy on account of their strictly geometrical orbicular circumference and their distinctly outlined clean-cut borders (Fig. 162). This feature at once attracts attention. It is altogether exceptional for the disk to be incomplete or

FIG. 162.—Spot of herpes circinatus trichophyticus on a child's neck.

the circle broken. When the affection is lodged on the fingers, the circle is continued from one finger to the other, just as at the eye or mouth it passes from one eyelid or one lip to the other.

The spot is pink or red, often with a dusky center, covered with powdery, flaky or crusted scales; it is marginate or plainly circinate through healing from the center.

Another valuable but inconstant characteristic is the *vesiculation* noted at the border of the spot; vesicles may be observed over the entire lesion, or there may be several concentric circles of vesicles (up to 7 on a cast in the Saint Louis Hospital). Sometimes, the vesicular contents are turbid or purulent. The vesiculation is of the eczematous type.

The floor of the spots is sometimes more or less edematous or infiltrated. The downy hairs may be dull and brittle or encased in scales at their base. Itching is very variable in its severity.

All these variations are much more dependent upon the kind of trichophyton involved than upon individual or accessory factors. As a rule, all the spots are of the same type in a given case.

The *course* of herpes circinatus is almost always rapid; in a few days, a spot will reach the dimensions of a silver quarter; the extension of the borders may be more than 1 mm. daily for some species. Slightly older patches may reach the size of the palm or the entire hand. Very frequently these spots are multiple and of different ages, the most recent spots being the result of re-inoculations. It was formerly taught that the confluence of two or more lesions may give rise to polycyclic spots or arabesques and that a considerable portion of the integument may become invaded; in all probability many cases of this kind belong to the group of epidermophytosis.

The *diagnosis* of herpes circinatus is often obvious at once through the clinical features; in doubtful cases, it will be confirmed by the microscopical examination of the scales from the periphery of the lesion. The parasite here appears in the form of band-like, slightly wavy, fairly regular, often bifurcated mycelium filaments, composed of short members (Fig. 161); even unstained specimens show the very close partitions between the square or rectangular segments, which is not the case in the mycelium of epidermic microsporia.

Confusion of herpes circinatus must be avoided with the trichophytoid patches of common eczema, which are less regularly outlined and accompanied by scattered eczematous lesions; with the eczematides, whose configuration is less orbicular and whose course is slower; with pityriasis rosea of Gibert, which Kaposi persistently classified under "herpes tonsurans maculosus," but which is distinguished by is course, its ovaloid patches and the absence of vesiculation; it is only the primary patch which may sometimes require microscopical examination.

The distinctive features differentiating the cutaneous trichophytoses from epidermophytosis will be discussed presently. Under certain special circumstances, the question of a differential diagnosis from various exotic epidermomycoses may arise.

Treatment by painting with pure tincture of iodin, repeated three times at intervals of two days, or with $\frac{1}{5}$ diluted iodin tincture in daily applications; or with iodinized vaselin 1 to 100, leads to a cure of herpes circinatus in from ten to fifteen days. This medication has replaced the formerly classical employment of turpeth-mineral salve 1 to 15 and of naphthol ointments.

Epidermophytosis Inguinalis (Eczema Marginatum of Hebra).—The cutaneous affection described by Fr. Hebra, under the name of *eczema marginatum* was recognized as of mycotic origin by Köbner, Pick and Kaposi, but continued to be confused with the cutaneous

trichophytoses. The accurate investigations of Sabouraud (1908) have demonstrated that it is always due to the same parasite, the *epidermophyton inguinale;* although very closely related to the trichophytons, it differs from these botanically and through the fact of its never invading the hairs.

The eczema marginatum of Hebra has its seat of predilection at the inner aspect of the root of the thighs. It begins in this region on one or both sides, as one or several nummular spots of a bright red color, sharply outlined and pruritic; by their rapid growth, these spots become confluent in circinate patches, with red circinate borders, often finely vesicular or bordered by white lamellar scales. Meanwhile, the center usually fades or remains pigmented, interspersed with scanty scales and excoriations, or with small crusts due to scratching. Around the principal patch, secondary patches often originate on the thighs, as far down as the knees, on the pubis or the buttocks, taking the same course and sometimes involving very extensive surfaces. On the scrotum and penis the lesions are less distinct, almost obliterated. The eruption may spread to the axillæ, the submammary folds, the legs and feet; not infrequently it coincides with epidermophytosis of the extremities; unless properly treated, it may last for months and years.

Not uncommonly, especially in men and more particularly in the well-to-do classes, eczema marginatum is contagious through sexual intercourse and also indirectly through water-closets or underwear; as a matter of fact, small epidemics are observed in colleges, workshops, asylums, etc. [The "jock-strap," the hired bathing suit, etc., are common sources of infection.]

Microscopical examination of the scales—which must always be taken from the red border—shows an abundance of mycelium composed of quadrangular or sometimes ovoid elements, resembling that seen in Fig. 161. Cultures grown on test media are radiating, powdery, of a lemon yellow color, or downy through pleomorphism.

Eczema vulgare is distinguished from epidermophytosis by its frayed margins and its less restricted localization; intertrigo, by its symmetry and its diffuse margins; erythrasma, by its relatively stationary behavior, its dry finely scaly surface, of a uniform dusky pink color, without vesiculation of the margins; the mycelium of the microsporon minutissimum is infinitely more slender than that of the epidermophyton (compare Fig. 161 and Fig. 166).

Epidermophytosis is differentiated from herpes circinatus trichophyticus by its predilection for the covered regions, by its tendency to become confluent in polycyclic surfaces or in arabesques; the patches are not invariably disks or closed circles, but are sometimes open on one side. The demonstration of the invasion of the downy

hairs, or cultures of the parasite, may be required in support of the diagnosis, but the matter is of no particular importance.

Treatment by painting daily with iodin tincture diluted with three or four parts of 80 per cent. alcohol, is usually sufficient; if necessary, it may be supplemented by the employment of a zinc salve with oil of cade (5%) and salicylic acid (1%). [Whitfield's ointment, Ac. Salicyl. 1.0, Ac. Benzoic. 2.0, Petrolati, 15.0 is most valuable.] Chrysarobin ointments are also very effective, but involve the risk of a severe and painful dermatitis.

Epidermophytosis of the Extremities.—The same epidermophyton inguinale—as discovered by Sabouraud in 1910—is the pathogenic agent of a very common affection of the interdigital spaces of the feet, which was formerly confused with eczema, dysidrosis, intertrigo, or maceration due to hyperidrosis of this region.

The lesions chiefly occupy the floor of the interdigital folds, especially between the fourth and fifth toe and the flexion folds of the toes. A macerated horny layer is present and becomes detached in the form of white shreds, leaving a bright red surface or a cheesy material; in the circumference a few vesicles are sometimes seen, which may dry or coalesce into an eczematiform surface. This dry, vesicular or impetiginous parasitic eczema may extend to the dorsal aspect of the foot as well as to the sole, even encroaching upon the plantar arch as far as the heel. With remissions and exacerbations the disease may be prolonged for many years or indefinitely. Through the itching induced by it, the burning sensations and superadded pyodermatitis, it becomes a source of distress, interferes with the wearing of shoes and may make prolonged walking impossible. Failure to recognize this dermatosis has often caused the patients to be subjected to strict diets, cures in watering-places, etc.

On the hands and fingers, epidermophytosis is likewise met with, although much less frequently, in the form of vesicular lesions or squamous, rarely circular exfoliations of the palmar surfaces; it is almost invariably confused with a dysidrosis.

Epidermophytosis of the extremities is accordingly eczematiform or dysidrotiform rather than like the palmar and plantar trichophytosis. Microscopic examination is necessary to establish a positive diagnosis; the mycelium is abundantly present in the scales. Cultures are difficult on account of bacterial contaminations.

Treatment.—The treatment is the same as that of inguinal marginate eczema, but in view of the thickness of the horny layer, the action of the dilute iodin tincture must be reinforced by moist dressings during the night, followed by careful scraping and cleansing. Sabourand recommends an ointment of lard and chrysarobin (1 to 3 to 100) with salicylic acid (5 to 100), avoiding all washing with soap and water. [In my experience, a five-day course of Dreuw's ointment is incomparably the most effective treatment.]

Microsporia.—The parasite of tinea tonsurans with small spores, the microsporon (p. 421), was discovered by Gruby, then misinterpreted, lost, and re-discovered by Sabouraud in 1892. It differs from the trichophytons in its microscopical and botanical features and in the appearance of the lesions produced by it. The contributions of Adamson, C. Fox and Blaxall, Bodin, etc., and especially the work of Sabouraud, collected in his book on The Tineas, 1910, have definitely settled the scientific data concerning it.

The *microsporon Audouini*—the first discovered, which infests especially Northeastern Europe, is at the present time endemic in England, causes two-thirds of the cases of microsporic tinea tonsurans in Paris and is responsible for the epidemics in children's asylums and schools—is now known to be not the only species met with in man.

There are about ten other microsporons which are for the most part of direct or indirect animal origin (*m. lanosum* or *caninum*, *m. felinum*, *m. equinum*) and give rise in nearly all countries of the earth to sporadic cases or small family epidemics.

The microscopical demonstration and the culture of microsporons are the same as those of the trichophytons. Cultures of the microsporon Audouini, on the maltose medium of Sabouraud and at room temperature, become visible on the fifth day, measure 5 cm. at the end of a month and resemble a layer of white or grayish short-nap wool. Cultures of microsporons of animal origin are of quicker growth and more downy, their fluff showing abundant multilocular spindles under the microscope; furthermore, the last-named cultures undergo from the fifth week on a downy pleomorphic degeneration, not shown by cultures of the microsporon Audouini.

Microscopical examination of microsporon cultures shows modes of reproduction akin to but different from those of achorions and trichophytons, in the form of pyriform swellings on the filaments, large spindle-shaped and partitioned conidia, pectinate hyphens, clusters of cylindrical and sessile conidia, etc.

The microsporon Audouini affects exclusively the scalp of children, where it causes the *microsporic tinea* which has been described elsewhere in the work (XX).

It very rarely vegetates on the hairless skin, where it produces scaly pinkish spots of ephemeral character and self-limited duration.

Animal microsporons outside of the hairy scalp and even in adults sometimes cause erythemato-squamous, nummular or circinate, pink or dun-colored spots, in large numbers, exceptionally even generalized over the entire body. These spots of cutaneous microsporosis may last a month and a half. A few cases of microsporic sycosis of the beard have been observed in men.

In the scales of tinea tonsurans with small spores, or in those of

epidermic microsporosis, examination in potash solution shows abundant twisted and serpentine filaments, with apparently few partitions, frequently bearing small lateral protuberances. After staining, this mycelium appears divided into septa at short intervals.

Tropical Epidermomycoses.—Tokelau or Tinea Imbricata.—This dermatosis is endemic in the islands of the Pacific and in Indo-China. It is observed in natives of all ages.

The disease, which persists throughout life, manifests itself as regular erythemato-squamous concentric circles. The shell-like scales are adherent at their peripheral portion, loose at their central portion and overlap like the tiles of a roof. The circles and their underlying patches increase in number and size, become interrupted through a variety of factors, until the entire integument becomes covered with squamous arabesques, including the face, the extremities and the nails, but not including the scalp. The patient roughly resembles a case of ichthyosis; he suffers from severe and continuous pruritus.

The microscope shows a dense network of branching and spore-bearing mycelium in the scales; the condition is referred by Manson to a trichophyton capable of inoculation from man to man but not growing in cultures, namely the *trichophyton concentricum* of Blanchard; Tribondeau describes the organism as *aspergillus lapidophyton*. Castellani reports successful cultures and inoculations of *endodermophytons*.

Carates and Central American Epidermomycoses.—Under the name of *carates, mal del Pinto,* or *Pinta,* are designated numerous epidermomycoses due to chromogenic parasites, widely distributed in tropical America and although known for a long time, as yet inadequately studied. They are characterized by spots which become confluent in large desquamating lobulated patches and may become generalized. There exists a black, blue, violet, red, yellow and a white variety; all, except the red one, may ultimately turn white. The odor is offensive, the hairs fall out, the nails remain intact. Association with scabies is common. The scales enclose a profuse tangle of mycelium. The chromogenic parasites, studied by Montoya, belong to the groups of aspergillus, penicilium, monilia, montoyella, microsporon and trichophyton.

Aside from these carates, various imperfectly known epidermomycoses are met with in Central America, which give rise to red patches covered with white scales, sometimes hyperkeratotic, with polycylic contours; they may attack the nails and are of indefinite duration. In a case (Fig. 163) observed by me in a patient from Ecuador, published in 1903, the scales enclosed a network of a mycelium which Bodin (of Rennes) successfully grew in cultures; he found it to be related to trichophytons and still more closely to

the lophophyton gallinæ, the parasite of chicken-favus, which causes the "white comb" in fowls. There are probably more or less analogous epidermophytoses due to various fungi in many tropical countries.

The *treatment* of these exotic mycoses is extremely tedious. I have obtained the best results with chrysarobin in ointments (of 1 to 3 per cent.), or in varnishes like traumaticine.

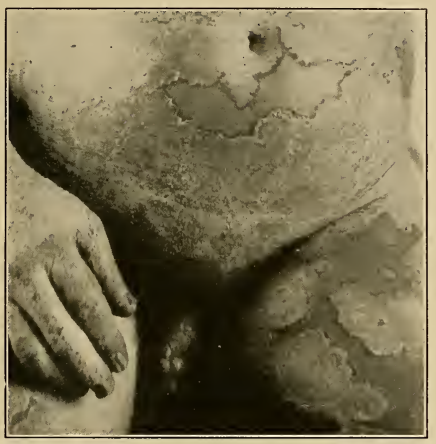


FIG. 163.—Central American epidermomycosis of seven years' standing, diagnosed as carate; note the lesions of the nails and genital organs. The patient suffered at the same time from scabies.

Pityriasis Versicolor.—Pityriasis versicolor is a parasitic affection of the epidermis, characterized by yellowish and scaly spots, caused by the *microsporon furfur* or *malassezia furfur*.

Confused by the ancients with pigmentary spots, pityriasis versicolor was distinguished from these by Willan, who classified it among the squamous diseases and gave it the name it bears. Eichstedt, in 1846, discovered the parasite, which was christened by Ch. Robin.

Pityriasis versicolor manifests itself as sharply defined patches, varying in color from a dirty yellow, tan or chamois to a dark brown, sometimes with an admixture of a pinkish shade. The color varies in different persons, different regions, and even on the same spot at different times; hence the name of *versicolor*. In the colored races, the spots are lighter than the normal skin and achromic by contrast.

The spots are slightly scaly, powdery or entirely smooth. Their

essential feature, however, is that the horny epidermis is here less adherent than under normal conditions, so that on vigorous scratching with the nail a desquamated shred becomes detached without bleeding. This "*nail-scratch* or *flake sign*" is almost pathognomonic and is due to the fact that the presence of the parasite permits the superficial horny layers to slide over the deeper layers.

The spots are not prominent. Their shape and dimensions are extremely variable. They may be punctate, guttate, in disks, rings or patches, or cover large surfaces with geographical contours, sometimes involving a large portion of the thorax; these various configurations are often seen on the same individual.

Any region of the integument may be the seat of pityriasis versicolor, with the exception of the hands and feet. Its seat of election is the upper part of the chest, in front and behind, whence it reaches the shoulders, the flanks, the abdomen, the groins, the arms and rarely the legs. It is exceptionally observed on the neck, the chin and even on a large portion of the face.

The growth of the vegetating parasite as a rule causes no itching at all, so that the patients are not aware that they are affected with a parasitic dermatosis. The duration of the disease is always very protracted and indefinite; it may, however, heal spontaneously, for it is never met with in the aged. It progresses and becomes aggravated under the influence of sweating and in the absence of ablutions and remains stationary under contrary conditions.

Microsporon furfur grows exclusively in the horny layer and produces no inflammatory reaction. It never invades the hairs. In the shreds detached with the finger-nail may be seen (after clearing with potash and under a magnification of 300 diameters) numerous clusters of fifteen to thirty round spores, with double contours, measuring from 3 to 5 μ. From these masses, radiating and interlaced mycelium filaments which traverse the intermediate spaces are given off. They are winding, short, somewhat irregular, slightly branching, divided by septa and in places spore-bearing (Fig. 164).

The culture of microsporon furfur, remarkably enough, meets with great difficulties; successful cultures were grown by Matzenauer and Nicolle, the latter using glycerin-agar.

The *etiology* naturally depends upon the transmission of the parasite. But pityriasis versicolor is very slightly contagious; it is rare for husband and wife to infect each other or to convey the contagion to their children. Köbner after many attempts succeeded in inoculating himself and in infecting rabbits. The incubation lasts over a month.

This epidermomycosis is common among adolescents and adults of both sexes; it is very rarely seen at an early age. In all probability it requires special regional conditions. The influence of a

tuberculous soil has been exaggerated; for some time, a relation between the microsporon and the agent of tuberculosis was erroneously surmised. The frequent coincidence is probably accounted for by the sweating of consumptives, the habitual wearing of flannels and the fear of bathing.

The *diagnosis* is based essentially upon the "flake sign;" a microscopical examination is almost superfluous. If only the physician will think of looking for this sign, a confusion with congenital pigmentary spots, chloasma, pigmentary syphilides, eczematides, etc., is impossible. The distribution of certain pigmentary spots of leprosy may be analogous to that of pityriasis versicolor.

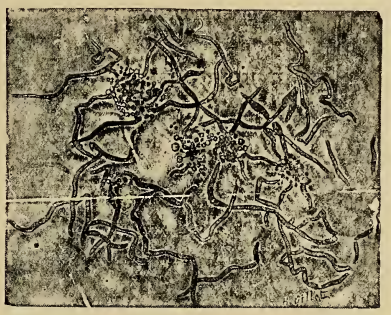


Fig. 164.—Microsporon furfur in a scale from pityriasis versicolor; stained with Sahli's blue. × 325.

Although common pityriasis versicolor may occur, as I have myself seen, among the inhabitants of Annam and Cambodia, there is in addition a *tropical pityriasis versicolor,* or *tinea flava,* or *parasitic achromia with estival exacerbations* (Jeanselme). This epidermomycosis is very common in the far Orient and is characterized by yellowish, slightly scaling spots which preferably occupy the face, neck and upper trunk, is of very slow development and difficult to cure. It is caused by *malassezia tropica* (Castellani, 1905) which has a short, swollen or club-shaped mycelium with spores not always arranged in clusters.

The *treatment* of pityriasis versicolor must aim at removing the horny layer which alone harbors the parasite. Not a spore must be left behind, so as to guard against recurrence; on the other hand, unnecessary irritation must be avoided. Tincture of iodin, employed as in herpes circinatus, lard with chrysarobin (1 to 1000), or simply soft-soap rubbed on for ten minutes followed

by a bath, are usually sufficient. For regions where the skin is sensitive, in the groins or in the axillæ, recourse may be had to naphthol (1 to 30) or sulphur (1 to 20) salves and to salicylic acid ointments (1 to 30), not forgetting washes with soap and baths, as well as disinfection of the body linen. [A simple and effective treatment consists in a daily bath with soap followed by a sponging with a 1 to 8 solution of photographers' *Hypo* for a week; the course of treatment, whatever it may be, should be repeated after two weeks' interval.]

Erythrasma.—The practitioner is usually not so well acquainted with this disease, although it is as common as pityriasis versicolor; it is an epidermomycosis due to the *microsporon minutissimum* and appears in the form of brownish or yellowish-pink patches especially in the inguinal folds.

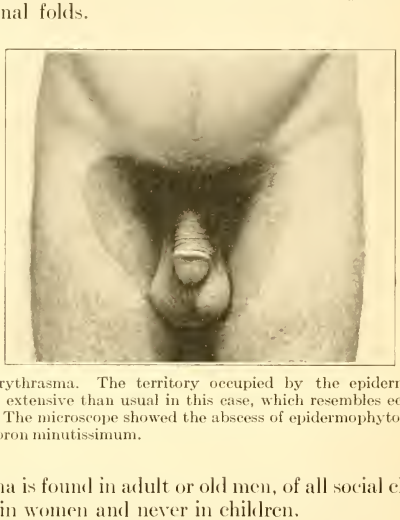


Fig. 165.—Erythrasma. The territory occupied by the epidermomycosis was somewhat more extensive than usual in this case, which resembles eczema marginatum of Hebra. The microscope showed the abscess of epidermophyton and the presence of microsporon minutissimum.

Erythrasma is found in adult or old men, of all social classes, much more rarely in women and never in children.

Its origin always escapes attention; the patients are usually unaware of having it and are ignorant of its onset. Subjective symptoms are absent; sometimes moderate itching is noted after sweating. The course is very slow; the duration is indefinite.

The seat of election of this dermatosis is in the genitocrural fold high up on the internal aspect of the thigh (Fig. 165); it rarely encroaches upon the pubis and the scrotum. The lesions are unilateral or bilateral. Much more rarely the axillæ are also involved.

The erythrasma patch, ordinarily from 5 to 12 cm. wide in all diameters has an always distinct, polycyclic and serrated circum-

ference; outside of the principal patch, small islands of the same character may be found on the thighs, the abdomen and elsewhere, but this is altogether exceptional. The color is uniform as a rule but very variable according to the moment when seen, dark red, deep yellow, brownish with a tinge of pink. The surface is level, powdery, finely squamous, and often criss-crossed in very fine folds. It is oily and moist during perspiration.

Examination of the scales under the microscope, with a magnification of 400 diameters, after treatment with potash solution, serves to demonstrate the parasite in some cases, but not easily. It is better, after removing the fat with ether, to stain the scales with Sahli's blue or with phenol-thionine (Fig. 166).

Fig. 166.—Microsporon minutissimum in a scale of erythrasma. Stained with Sahli's blue. × 1000.

This parasite is the *microsporon minutissimum*, or *discomyces minutissimus*, discovered by Burchardt in 1859. It appears in the form of very fine filaments, less than 1 μ wide, twisted and branching, apparently fragmented and provided with spores at their extremities. These filaments are extremely abundant and constitute a real network between the horny cells. Culture of the discomyces is difficult; but Michele, Ducrey and Reale seem to have obtained it and were able to inoculate it on man.

Erythrasma is very slightly contagious; it is rare for husband and wife to be simultaneously affected.

Transmission possibly takes places through underwear, water-closets, etc.

The *diagnosis* is obvious in the majority of the cases, although sometimes difficult.

Intertrigo is more inflammatory and has no sharply defined contours. Eczematides are rarely so narrowly localized. Partial prurigo is highly pruritic and lichenized. Inguinal epidermophytosis is of a bright red color, is polycyclical and marginated, takes a rapid course and has a much larger parasite.

The *treatment* is analogous with that of pityriasis versicolor and inguinal epidermophytosis, but erythrasma is more rebellious. In a few weeks an apparent cure is obtained, but great perseverance is needed in order to avoid a recurrence. [The interrupted or discontinuous method of sterilization yields the best results in this as in many other infectious diseases. In erythrasma as in pityriasis versicolor one week of treatment should be followed by two weeks of rest, and several courses of treatment are usually necessary.]

CHAPTER XXVI.

INFECTIOUS DERMATOSES—PYODERMATITIDES.

In the following chapters (XXVI to XXIX) I shall discuss the infectious dermatoses whose parasitical agent is established, known and specific, or practically specific.

Some are referable to internal infections, such as syphilides, the rose-spots of typhoid fever, of some tubercular eruptions, etc.

Others are due to external infections, like the pyodermatitides, soft chancre, some cutaneous tuberculoses, Biskra boil, etc.

The following review, however, by no means exhausts the question of the part played by infections in skin diseases.

For many eruptions the causative infectious agent is still unknown (example: eruptive fevers); or it is not specific, in the sense that different microbic species may lead to the same syndrome (examples: purpura, ulcers, multiple gangrenes, elephantiasis); or it is secondary, superadded, perhaps accessory (examples: eczema, artificial dermatitides, etc.).

Finally, in many cases, the role of an infectious agent is theoretical, suspected or vaguely surmised (examples: polymorphous erythema, secondary and primary infectious purpuras, primary erythrodermas, warts, vegetations, pemphigus, acute nodosities, molluscum contagiosum).

According to the plan here adopted, these various syndromes figure only in the first part of this work, which is devoted to the morphology of skin diseases. In this part only pathological entities with an established infectious etiology are considered.

These infectious dermatoses, according to the nature of their pathogenic agent, may be classified as follows:

1. Infectious dermatoses caused by cocci: these are the *pyodermatitides*, to be discussed first of all.

2. Dermatoses caused by bacilli, or *infectious bacillary dermatoses* (XXVII).

3. Infectious dermatoses caused by vegetable parasites of an order higher than the cocci and bacilli: *dermatomycoses* (XXVIII).

4. *Infectious dermatoses due to protozoa: spirochetes, leishmanias,* etc. (XXIX). This group comprises *syphilis* and a few more or less analogous exotic dermatoses.

PYODERMATITIDES.

The acute inflammations of the skin due to the ordinary microbes of suppuration or pyococci are called pyodermatitides.

Pyococci.—The vast majority of suppurations of the skin are caused by two groups of microbes: *staphylococci* and *streptococci*. In each of these groups numerous varieties or species are distinguished which are interpreted by some bacteriologists as simple strains. These varieties differ from each other in their morphology, their cultural characteristics, their habitat and—what is especially of clinical interest—in their virulence.

Staphylococci are cocci of somewhat variable size, which appear in pus in the form of diplococci, or in a series of three or four granules; in cultures they are arranged in masses or clusters.

They grow readily on most of the ordinary artificial culture media, preferably at 37°; they have a variable chromogenic capacity which is the basis of a differentiation between the cocci obtained from the pus, as follows: *Staphylococcus aureus*, *Staphylococcus albus*, *Staphylococcus citreus*, etc.; they liquefy gelatin more or less rapidly; they possess a high degree of vitality.

Nearly all staphylococci are pyogenic; on inoculation they produce subcutaneous abscesses, peritonitis, etc., in the majority of animals. When injected into the ear-vein of rabbits, they give rise, according to dosage and virulence, to a rapidly fatal septicemia, miliary abscesses of the kidneys, arthritis, osteomyelitis, endocarditis, etc. Garré, Bockhart and Rodet produced folliculitis, impetigo, furuncles, etc., by rubbing cultures upon the human skin.

Up to ten varieties or species of staphylococci have been described. One of great importance on account of its abundance on the skin is designated under the names of *Staphylococcus cutis communis*, or *Coccus polymorphis* of Cedercreutz. This microbe, which undoubtedly corresponds to the morococcus of Unna, presents itself in the scaly crusts of eczematides under the aspect of mulberry-shaped clusters; it grows on glycerin-agar in porcelain-gray streaks or drops and gives off a butyric odor. It does not liquefy gelatin. It is usually considered as non-pathogenic, but its inoculation on the human skin has been known to cause folliculitis or small vesicles of eczema.

Staphylococci are widely distributed in the air, in dust and in water; they are regularly present in the mouth, at the natural orifices, and are disseminated on the skin.

Streptococci are cocci arranged in small chains of variable length, often composed of diplococci; the size of the granules varies. Streptococci will grow on all ordinary culture media, preferably under anaërobic conditions; the optimum temperature is

37°. They do not liquefy gelatin and form flakes in broth-cultures; their vitality is readily destroyed by the air. In order to isolate a streptococcus contaminated with other cocci, Sabouraud recommends growing the first culture in a pipette in bouillon or ascites-fluid; or, according to a familiar procedure, the suspected material may be planted on several slanting tubes of peptone-agar and numerous streak-cultures planted without recharging the platinum needle.

The virulence of different streptococci varies to an enormous extent. The *streptococcus* of *erysipelas*, discovered by Fehleisen, the *Streptococcus pyogenes* of Ogston and Rosenbach and the streptococcus of *puerperal infection*, undoubtedly belong to a single species. As regards other varieties, differing in their morphological and biological features as well as their habitat, the question has not yet been settled.

On inoculation into a rabbit's ear, a very virulent streptococcus gives rise to fatal septicemia; a less virulent germ causes erysipelas; a still weaker one will produce an abscess. By means of intravenous injections into animals, practically all the affections caused by streptococci in human beings have been successfully reproduced.

Streptococci have their ordinary habitat in the air, in the ground, on the skin and especially in the mucous cavities. They are less frequent on the epidermis than staphylococci, but are constantly present in the mouth of healthy individuals.

The relations existing between the Streptococcus pyogenes and the Streptococcus of the saliva (Veillon), the Streptococcus of scarlet fever, the small granular streptococcus, those which liquefy gelatin or are encapsulated, etc., have not been definitely established. Up to 46 species have been described (Gros).

Other microörganisms in large number, but not properly speaking pyococci, are likewise more or less commonly encountered on the skin and in the surroundings of the natural orifices, such as: various *micrococci*; the *bacillus of seborrhea*, *epidermidis*, *cutis commune*, *subtilis fluorescens*, *coli*, *pseudodiphtheriticus*, the *bottle-bacillus* or *Malassez spores*, etc.; furthermore, a *sarcina*, a *leptothrix*, various *saccharomyces*, etc.

The germs of the skin vary enormously in number according to circumstances. The average is assumed to be 40,215 per square centimeter; from 85 millions to 1212 millions of germs are left in a bath, according to Remlinger; they flourish with especial luxuriance in the moist and hairy regions of the body. Sabouraud has shown that they are more abundant around pathological lesions of any kind. Their principal receptacles aside from the mucous orifices are the follicular funnels which constitute both a shelter for the germs and the weak point in the epidermic armor (Chapter XIX).

It must be understood that no scrubbing or ever so careful and prolonged disinfection can be relied upon to remove or destroy all the microbic inhabitants of the epidermis. With special reference to pyodermatitis, it may be said that the seeds are practically ubiquitous. However, the absence of cleanliness, contact with contaminated individuals, with soiled clothing or other articles, will naturally greatly increase the chances of infection.

Pathogenesis.—In this respect, four points require attention: the portal of entry, the virulence, the resistance of the soil and the pathogenic mechanism.

A. The *portal of entry* of pyogenic microbes is external in the vast majority of the cases. It is only in the pyemias, etc., that they reach the skin by the vascular route, giving rise to intra- or subdermic abscesses.

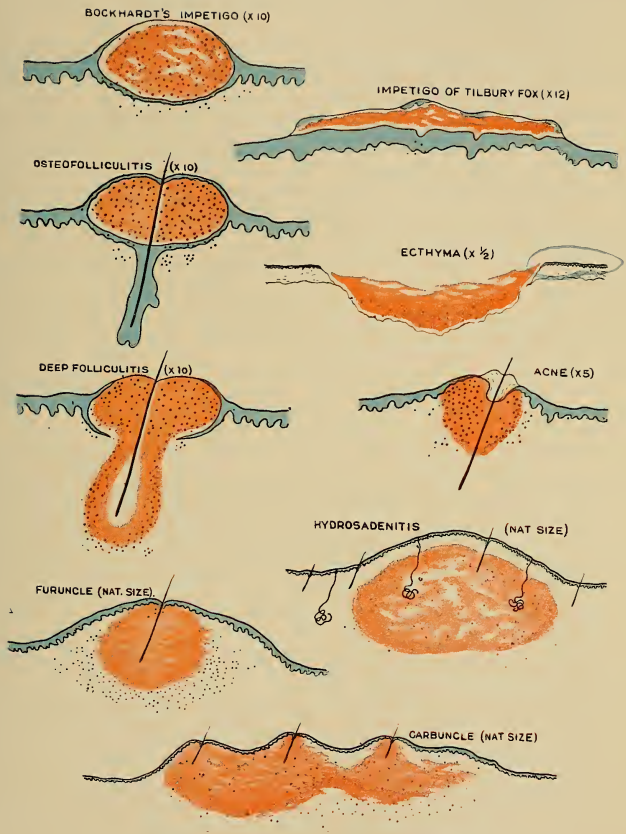
The protective barrier formed by the horny layer is removed through traumatisms of all kinds, by parasitis such as sarcoptes or lice and by scratching in case of pruritus. It is likewise impaired by maceration or chemical lesions of the epidermis, such as those caused by poultices, plasters and strong antiseptics like bichloride of mercury, carbolic acid, etc. It is noteworthy that the latter are usually more injurious for the epidermis than for the germs contained in it, thereby favoring the propagation of the pyodermatitis. The present tendency therefore is to replace the antiseptic dressings, which were so popular in the past, by aseptic dressings.

The reason why the follicular orifices so often become the portal of entry of infection is that they are usually already inhabited by germs and on the other hand are easily damaged by friction, movements, etc. Preëxisting lesions of the epidermis, eczematization, vesicles and bullæ, offer a ready-made culture-medium for the pyococci; secondary infection, superadded suppuration or impetiginization is therefore extremely common.

It is often difficult to estimate the relative significance of the primary cause and of the secondary infection, as a matter of fact, various chemical substances and certain specific microbes may be pyogenic in themselves.

B. The degree of *virulence* of the parasites, pyococci and others, is extremely variable, not only according to the species or strain, but in the same strain. Some behave like simple saprophytes; others again are highly pathogenic. The virulence of a microbe may be attenuated or exalted by different laboratory procedures. Clinically, it is governed by conditions not all of which have been established; the best known although not the only cause of increased virulence is primary growth in a pathogenic focus, the effect being comparable to the well-known increased virulence by passage through an animal; it accounts for the auto-inoculations, the contagiousness

PLATE II



Schematic Comparison of the Pyodermatitides.

Pus and necrotic tissues are shown in yellow; the dots indicate approximately the relative abundance of polynuclears in the epidermis and in the cutis. The amplification is stated with the name of each variety.

and persistence of some pyodermic lesions. A very considerable number of common microörganisms are actually under suspicion, being capable of assuming pathogenic properties under a variety of circumstances.

C. The *resistance of the soil* against pyococci is also extremely variable. Although immunity against them, either temporary or permanent, does not exist, the susceptibility is evidently increased under certain conditions. In children and youthful individuals, such conditions are represented by the so-called lymphatic constitution or scrofulous diathesis, by undernutrition, general weakness and convalescence from disease. In adults, overexertion, general nutritional disturbances, such as diabetes and cachexia, likewise create a predisposition for pyodermatitides. The part played by local circulatory disturbances or nutritional disturbances of the skin manifests itself for example in varicose limbs. These factors explain not only the easy onset of superficial pyodermatitides but also their multiplication, their liability to recurrence, their obstinate character and the tendency to complications with lymphangitis, abscesses, adenitis and to termination in the grave forms of staphylococcia and streptococcia which may lead to death in weakly children and cachectic individuals.

D. Staphylococci and streptococci both possess, undoubtedly due to their secretions, a vasodilator action and an attraction for leukocytes known as *positive chemotaxis*. Under their influence the skin becomes congested and the white corpuscles swarm into the cutis, or, according to the case, into the epidermis, together with a certain amount of plasma. Certain differences are noted, however, between the properties of the two groups of pyococci.

The staphylococci attract especially the polynuclears and produce the smooth, creamy, thick pus which was formerly described as "laudable pus." The streptococci are in a general way less pyogenic; they give rise rather to an outflow of plasma and the secretion of a turbid serous fluid or of thin serous pus. However, according to Jadassohn, staphylococci may also give rise to serous bullæ. The Staphylococcus aureus, moreover, possesses necrotizing properties, as shown by the core of furunculus and the slough of carbuncle. Mixed infection produces intermediate effects.

Clinical Forms.—A series of pathological types is referable to the penetration of pyococci into the epidermis, into the pilosebaceous follicles, or into the cutis. I have endeavored to convey an idea of the principal types in the subjoined schematic figures (see Plate II).

Among the *staphylococcic* pyodermatitides of external origin, I have already described Bockhardt's impetigo, impetiginous eczema, ostiofollicular pustules and deep folliculitis, sycosis, etc. Furunculus, carbuncle and hidrosadenitis will be discussed further on.

Abscesses, diffuse phlegmons [cellulitis] and suppurating wounds belong to the domain of surgery.

Staphylococci brought to the skin by way of the blood give rise to multiple and successive emoblic abscesses, as one of the manifestations of pyemia, which is accompanied by grave general disturbances.

I have previously had occasion to state that the miliary abscesses of little children are not referable to the last named pathogenesis, as formerly believed to be the case, but that they are the result of an ascending infection of the sudoriparous canals.

I repeat that an association of various pyococci may be held responsible for impetigo vulgaris and ecthyma (pp. 165, 169), for various ulcers (XV), probably for pustular acne (p. 386), the microbic eczemas (p. 66) and the eczematides (p. 97), as well as for impetiginous onyxis (p. 434).

Referable to cutaneous *streptococcia* are: the impetigo of Tilbury Fox, ecthyma, leg ulcer, erysipelas (which is not usually considered in the scope of dermatology), the lymphangitides, lymphangitic abscesses, phlegmons and adeno-phlegmons, which belong to the domain of surgery.

Perlèche.—Perlèche—a very trifling affection, interesting on account of its contagiousness and the diagnostic errors which it occasions—consists of a circumscribed redness of the two labial commissures, with maceration of the epidermis and often fissure-formation. It is frequent in children who contract it in school and transmit it to the family; it may last for weeks and months. Sometimes it coexists with impetigo vulgaris or impetiginous stomatitis.

Perlèche seems to be a simple regional streptococcia. By J. Lemaître, who described it in 1886, it was attributed to a so-called Streptococcus plicatilis, which is probably in no way specific.

A differential *diagnosis* must be made from herpes, which is rarely bilateral and always vesicular at the onset; and especially from mucous patches, but these are accompanied by other manifestations of secondary syphilis.

Perlèche is treated like impetigo. Silver nitrate, Alibour water, yellow precipitate ointment, or steresol, etc., will promptly check it. The children must not be allowed to kiss and their glasses and tableware should be washed in boiling water.

Finally, there occurs *non-suppurative* manifestations of the various pyococcias, such as certain *purpuras* (Chapter III), probably *botryomycoma* (p. 704) and perhaps the *necrotic* and *keloid* forms of *acne*, etc.

Furunculus.—A furuncle or *boil* is a massive folliculitis with a usually very acute inflammatory course and necrotic character. Boils are due to the Staphylococcus aureus, as discovered in 1880

by Pasteur, who soon afterward was able to demonstrate the "microbe of furuncle" in osteomyelitis.

Whether it be derived from a contagion, or what is more common, from auto-inoculation of another staphylococcic lesion, the microbe must necessarily have been introduced rather deeply into the follicular canal in order to produce a furuncle. Hence, boils preferably develop in regions exposed to rubbing from clothes or implements of work; they are common in rag-pickers, refiners, mechanics, riders, etc. Fatigue, nervous disturbances, errors in diet and convalescence from acute diseases are predisposing factors. Diabetes mellitus especially constitutes such a favorable soil that the urine should be examind as a routine procedure in all cases of furunculosis.

In certain individuals, through the effect of a very marked predisposition or rather because immunization does not occur as under normal conditions, boils follow one another for a period of months and years. This furunculosis may be connected with a run-down condition, auto-intoxications, etc.; but its cause often remains undemonstrable.

A furuncle begins with a red acuminate elevation with a hair in its center; its two essential features are induration and pain. There is often considerable edema of the vicinity. In three to five days, the growing protuberance has assumed a purplish, later pustular apex. Having reached maturity, the furuncle softens and opens; after a flow of pus, a core or slough is eliminated, consisting of necrotic cellular tissue infiltrated with pus. The pain subsides as soon as the discharge of pus is freely established.

A furuncle always leaves a more or less visible cicatrix, unless it has been *aborted*, spontaneously or as the effect of treatment; in this case, the induration lasts from three to twelve days and finally becomes absorbed. In weakened or diseased persons, boils are accompanied by extensive edema, fever, malaise, etc.

Boils may be situated wherever there are hair follicles, but the site of election is at the nape of the neck; next in order follow the back, the buttocks, the lower limbs, the forearms, etc.

Furuncle of the upper lip gives rise to a severe and very alarming inflammatory tumefaction, and the possibility of lymphangitis, phlebitis and meningeal complications must be borne in mind.

Furuncles of the *auditory meatus* are noteworthy on account of the resulting pain, which is very severe, radiates widely, interferes with mastication and prevents sleep. These furuncles are often associated with auricular eczema; they have a tendency to recur and may affect one ear after the other.

A furuncle differs from ordinary suppurative folliculitis by the severity of the inflammatory reaction, the induration of the tissues, the pain and the *core* or slough. It is distinguished from hidros-

adenitis, which moreover is practically limited to the axilla, by its acuminate form and by its core. The differential diagnosis from malignant pustule [anthrax] may, in exceptional cases, prove somewhat difficult.

Treatment.—At the onset, abortive treatment may be tried; for this purpose, a finely pointed galvanocautery or thermocautery needle may be inserted in the center of the furuncle, or it may be painted twice, at an interval of twelve hours, with iodin tincture or iodo-acetone. The adjacent skin should be cleaned with an antiseptic soap and washed with camphorated or resorcinated alcohol.

In its developed stage, the furuncle must not be irritated by strong antiseptics, which would cause a complicating artificial dermatitis, without any benefit whatever. It suffices to make two daily applications of a hot spray with weak carbolic, resorcin, or phenosalyl water, or simply washing with Alibour water; the parts are then covered with cotton wadding, moist dressings with boiled water, or still better with a layer of cotton covered with borated starch glycerolate (1 to 10), which affords a better protection against neighborhood inoculations.

I have never found it necessary to incise a furuncle. It would be more justifiable to perform complete excision at the onset, provided the patient and the locality permit. The pain caused by interstitial injections, even with gaseous oxygen is not offset by their advantages. In the period of repair, it is advisable to cover the orifice with mercurial plaster or with yellow oxide of mercury salve or a piece of gauze.

[An attempt to abort a boil by the accurate application of a solution of iodin to its apex, repeated three times with twelve-hour intervals, should always be made at the inception of the process. It is useless when there is already a considerable induration. In cases of furunculosis every follicular inflammation, even the most insignificant, should be regarded as suspect and treated abortively. I agree with the author in condemning incision of a boil; it is bad practise, painful and useless. I earnestly recommend the following treatment: the affected area is washed with alcohol or 1 to 2000 bichlorid alcohol. The boil is then covered with a 3 to 4 cm. square of salicylic acid plaster made according to the formula of Dr. Klotz: ℞ empl. diachyli 60.0, empl. saponis 25.0, ceræ 2.0, vaselini 8.0, acidi salicylici 5.0, M. ft empl. secundam artem. This plaster mass rolled in sticks for convenience is spread as required, smooth, even and thin on cotton-sheeting (muslin) and applied over the boil. It is soothing, protective and sufficiently occlusive while not adhering firmly enough to cause retention of pus. It should be removed twice a day, the free pus very gently squeezed out, the surface washed with a mild antiseptic and fresh plaster applied. In

cases of furunculosis I employ the stock vaccines, four or five injections at intervals of three to five days.]

Internal treatment, with colloidal sulphur, sulphuric acid lemonade, sulphur compounds, hyposulphites, ichthyol, etc., is very variably estimated. Beer-yeast, preferably fresh, in doses of three tablespoonfuls daily, not infrequently produces a sudden subsidence of the inflammation and the pain. When it fails, beer-yeast from another source should be tried, or it may be replaced with a different ferment, for example a lactic ferment.

In furunculosis the vaccine therapy of A. E. Wright, even with a stock vaccine of Staphylococcus aureus, constitutes an excellent although sometimes unreliable method of treatment.

Care must of course be taken to correct the patient's hygiene; the dietetic regimen indicated in diabetes, auto-intoxication, arthritism, etc., may be called for in some cases. A cure at watering places with sulphur or arsenical springs is sometimes very useful in obstinate cases of furunculosis.

Systematic treatment of furuncle, furunculosis and other staphylococcias with pure metallic powdered zinc mixed with zinc oxide, in doses of 4 to 8 grams daily, as recently introduced by Frouin and Grégoire, is extremely simple and sometimes seemed to be efficient in my experience.

Carbunculus.—Carbuncle (French, anthrax) may be considered as an agglomeration of furuncles. The etiology is the same as that of furuncle. Garré successfully produced a carbuncle through inoculation of staphylococci derived from a case of osteomyelitis.

The lesions are more extensive than those of furuncle; the necrosis of the perifollicular cellular tissue becomes confluent in large areas which are finally detached by the suppuration and appear no longer in form of circumscribed sloughs but under the aspect of sometimes very extensive gangrenous shreds.

[The differences between carbuncle and furuncle are not simply a matter of degree. The phlegmonous inflammation in furuncle starting in a follicle always remains definitely circumscribed in a globular form, with the follicle as its center, though it may extend as far as the hypoderm. In carbuncle a similar infection extends into the hypoderm and then spreads to a greater or less extent laterally in the hypoderm ascending by the path of the *columnæ adiposæ* to the surface where it appears as a group of secondary furunculoid elevations in the neighborhood of the primary furuncle. A carbuncle is always a grave affection because it is capable of indefinite lateral extension in the hypoderm.]

The onset is often marked by chills, malaise, general prostration, anorexia, while at the same time a very hard red swelling makes its appearance, which is the seat of severe tearing pain. The neighbor-

ing edema is usually considerable and very extensive. At the end of four or five days a few small vesicles or pustules manifest themselves at the follicular orifices of the carbuncle; these become transformed into openings or craters from which pus escapes in increasing amounts and the grayish necrotic masses are subsequently evacuated. The skin between the orifices is purplish and swollen, often eroded; the bridges separating the orifices are often destroyed by purulent disintegration or necrosis. The openings of a carbuncle are accordingly multiple, irregular and winding. The period of evacuation lasts from a fortnight to a month. Spontaneous pain ceases as soon as the deep sloughing reaches its limit. Repair takes place by granulation; it leaves a very noticeable, often retracted and stellate cicatrix.

In weakened patients, or in diabetics, etc., the woody induration at the onset of a carbuncle may attain enormous proportions (*woody phlegmon of the aged*). In the period of suppuration, enormous winding cavities and purulent tracts are formed and sometimes large gangrenous patches. These *diffuse* or *malignant* carbuncles are accompanied by high fever, excitement, wild delirium or profound depression; they may lead to death through septicemia, hectic fever, or by rupture of large vessels or opening into large cavities.

The *prognosis*, which is very variable, would seem to depend either on the virulence of the infection or on the character of the soil. The duration of a moderately severe carbuncle is from a month to six weeks.

The *treatment* of benign carbuncle is patterned on the treatment of furuncle. Incision is rarely necessary or advisable. In large carbuncles, the introduction of sticks of Canquoin's paste (therapeutic notes, § 11) evidently shortens the total duration of the process. The treatment of malignant carbuncle belongs to the domain of surgery.

Hidrosadenitis.—The hidrosadenitis of Verneuil, or *tuberous abscesses of the axilla* of Velpeau, are intradermic or subdermic abscesses, the size of an almond to that of a large nut or more, which occur exclusively in the axillary region.

These abscesses are more common in women, developing especially in cases of intertrigo, lack of cleanliness, hyperidrosis, but principally of axillary eczematides.

One or more indurations develop, usually without general disturbances, with a sensation of pruritus, tension and distress. These indurations are perceptible on palpation, which is painful; gradually they form a demonstrable, hemispherical, red, non-acuminate prominence.

They may undergo absorption, but more frequently they soften and open through the thinned and reddened skin, emptying a

creamy pus, but usually no slough. The development of each nodule lasts from ten to fifteen days; they succeed each other, sometimes in both axillæ, for weeks and months. The affection has a tendency to recur each summer. Complications are rare.

On account of the differences in appearance and course, as well as the usual non-coincidence of these tuberous abscesses with furuncles in other regions, they are believed to be due to a staphylococcus infection of the [large axillary] sudoriparous glands rather than of the pilosebaceous follicles.

The treatment is the same as that of furuncle. The hairs of the region should not be cut or shaved. I have known beer-yeast or various ferments work wonders in cases of protracted duration; the same remark applies to the modern treatment with zinc. Vaccine-therapy is also indicated and sometimes very successful. Scrupulous cleanliness is required to guard against recurrences.

CHAPTER XXVII.

INFECTIOUS BACILLARY DERMATOSES.

The infectious dermatoses caused by bacilli are: cutaneous tuberculosis; leprosy, glanders, malignant pustule or anthrax, cutaneous diphtheria, soft chancre and probably also the verruga [Peruana] of Carrion and rhinoscleroma.

A number of other affections have also been attributed to specific or non-specific bacilli, pure or associated, for instance, with spirilla (fuso-spirillary symbiosis), as follows:

Wound-diphtheria and hospital gangrene, which belong to the domain of surgery; tropical phagedenic ulcer (p. 301, ulcero-membranous stomatitis (p. 306), noma (p. 308) and balanoposthitis erosiva circinata (p. 311).

TUBERCULOSIS.

The cutaneous manifestations of tuberculosis constitute at the present day an important chapter in dermatology. This chapter has only gradually been built-up and had no scientific basis before the discoveries of Villemin and Koch.

Thus lupus vulgaris which had been distinguished from the other dermatoses by Willan and Bateman, was named *tuberculous* only because its characteristic lesion belongs to the class of tubercles, in the dermatological sense of this term. Clinicians—Lugol, Bazin, Hardy, Lallier, Vidal and Besnier—grouped it among the *scrofulides* and more or less clearly surmised its tuberculous character. This was demonstrated histologically by Friedländer and others, experimentally by Max Schüller, Leloir and especially by R. Koch.

The evolution of our views in regard to the other types of cutaneous tuberculosis has undergone analogous changes.

Among the features on which a diagnosis of a cutaneous tuberculosis is based at the present day, only two have a positive scientific value, namely:

1. The presence of Koch's bacillus in the lesions.

2. The positive outcome of inoculation of the affected tissue into animals, especially guinea-pigs which are sensitive to this infection.

The other features permit merely a presumptive conclusion, although of considerable weight when several are present. Such features are:

1. A histological structure in conformity with that of known tuberculous lesions.

2. A positive local reaction to Koch's tuberculin A.

3. A certain clinical appearance and course and special etiological factors.

4. The coexistence of undoubtedly tuberculous manifestations.

A serodiagnosis of tuberculosis unfortunately is not possible.

Clinical Forms.—Five pathological types are known which in a general way satisfy these conditions and almost regularly present the six features enumerated above: *Tuberculous ulcer, tuberculosis verrucosa, tuberculous gumma, fungoid tuberculosis* and *lupus vulgaris*. I grouped these together under the generic heading of "*cutaneous tuberculoses*."

The contributions of the last thirty years have shown that there exists in addition a considerable group of dermatoses which without usually fulfilling the first two conditions so frequently comply with the four latter as to justify the surmise of a close connection existing between them and tuberculosis.

These dermatoses I have named *tuberculides* and the majority of modern authors interpret them, like myself, as attenuated tuberculoses. It is only the limitations to be made to this group that is still a subject of discussion.

It must be emphasized from the start that while this classification and these subdivisions are justified in the great majority of the cases and considerably facilitate their description and study, nevertheless numerous transition forms are met with between the various types of cutaneous tuberculoses as well as between these and the tuberculides. The existence of transitions between the various manifestations of the same cause is in no way remarkable.

General Etiology and Pathogenesis.—It would involve an encroachment upon the domain of general pathology to describe in this connection the Koch bacillus, to study its biological properties, its cultures, its secretions, its pathogenic properties in man and in animals, its portals of entry and the modifications apparently wrought in the course of the resulting disease through the properties of the affected territory. It is sufficient to emphasize the great extension of the field of tuberculosis through modern investigations. This infection at the present day appears to be infinitely more widely distributed than was formerly believed.

It is in reality essentially an infection of childhood. When it attacks the newborn, which is rare, the patients almost invariably die. In the course of late childhood, the number of infected individuals rapidly increases until, according to Naegeli, it reaches the proportion of 96 per cent. shortly after puberty. By no means all of these cases succumb to phthisis, meningitis or tuberculous septicemia; the majority resist the infection. When the initial lesions heal, various contingencies may arise: the tuberculosis may become

entirely latent, only the tuberculin reaction or possibly the autopsy being capable of revealing its existence. Or sometimes, so-called manifestations of local tuberculosis may persist or develop later on (glandular, bony, articular or cutaneous), constituting the "scrofula" of the older writers; furthermore recrudescences or the onset of new manifestations may occur at any time of life.

The influences which determine the course taken by the tuberculous infection are multiple and imperfectly known. One may incriminate the nature of the organic soil, the general hygienic conditions, the action of certain infections such as measles, etc., the effect of overwork, repeated pregnancies, etc., but the principal factor in recrudescences and in the development of new lesions undoubtedly is re-infection or superinfection of heterogenous or autogenous origin.

It has been experimentally demonstrated, however, that the re-infection of an organism invaded by the tubercle bacillus does not produce the same effects as the first inoculation, but different effects; this is known as the *allergy* of von Pirquet.

The subcutaneous inoculation of virulent Koch's bacilli into an animal of a very sensitive species, like the guinea-pig, produces at the end of ten or twenty days a nodosity which undergoes ulceration, *i. e.*, the tuberculous chancre; this is followed by glandular enlargement, then regularly by a generalized visceral tuberculosis affecting chiefly the spleen and the liver, from which the animal dies. Experiments on monkeys, in Java, in 1905, under the direction of Neisser, furnished practically identical results irrespective of the source of the tuberculous virus.

When an already tuberculized guinea-pig is re-inoculated with bacilli at another point of the skin, preferably between four and six weeks after the first infection, the immediate result is an ecchymosis and a slough which falls off leaving a wound which heals without corresponding glandular enlargement; this is known as *Koch's phenomenon* (1891) and constitutes a particular instance of allergy.

In animals of more resistant species, this allergy manifests itself differently and the same is undoubtedly true for man.

Returning to human cutaneous tuberculosis, with which alone we are here concerned, the following points must not be lost sight of: the individuals in which it is met with are practically all previously infected with tubercle bacilli; the re-infecting agent may be of exogenous or autogenous origin, according as new bacilli come from the outside or from a focus of tuberculosis borne by the patient himself; in the latter case, the cutaneous re-infection may occur through contiguity (derived, for example, from a subjacent bony or glandular tuberculosis), or it may result from a metastasis through the lymphatic route or the blood route.

These pathogenic conditions perhaps play a certain part in the diversity of aspect and course of the cutaneous lesions, although a smaller one than that pertaining to the existing state of allergy.

The question has arisen as to whether the origin and strain of the bacillus may not be concerned in the production of these differences. The bovine bacillus has occasionally been found, in cases of lupus for instance, more often in London and New York than in Paris; but the human bacillus is usually responsible. Burnet has noted some instances of human bacilli of attenuated virulence in cutaneous lesions. Investigations of this question are still under way.

At any rate, however, it must be admitted that although the reasons are not understood, tuberculosis of the skin manifests itself in lesions of very different form.

I shall review the principal, most common and most definite of these lesions, noting that they have been arranged on a diminishing scale of clinical malignancy and bacillary content.

Cutaneous Tuberculosis. — Tuberculous Ulcer. — This name is restricted to a primarily and essentially ulcerative lesion (p. 288) due to the Koch bacillus, which is abundantly present. This affection has also received the names of *miliary tuberculosis of the skin and mucous membranes;* and *acute tuberculous dermatitis.*

Ulcerated lupus, open tuberculous gumma, scrofula and fistulas derived from tuberculous osteitis, etc., do not fall under this definition.

It was in the mouth and notably on the tongue that tuberculous ulcer was first distinguished from analogous lesions by Ricord and subsequently studied by Julliard, Trélat, Féréol, etc. The observations of Coyne, of Jarisch and Chiari, showed that it occurred also on the integument under an identical form.

As a rule, tuberculous ulcer is observed only in already badly infected adults, notably in consumptives.

It has two seats of election: in the *mouth*, on the lips, tongue, the inner aspect of the cheeks, the pharynx, or in the circumference of the mouth and nostrils; or at the *anus* and its surroundings. It is rare on the genital organs, but may be found there and give rise to serious difficulties in diagnosis.

These localizations and the common preliminary of an open tubercular lesion of the lung, larynx, or intestines, in the same patient, are suggestive of the fact that tuberculous ulcer is generally referable to auto-inoculation of some fissure or erosion with bacilli from the sputum or the feces.

Sometimes, however, it is the result of contagion. I have observed it on the forehead of an apparently healthy woman, the wife of a consumptive. Cases such as those reported by Lehmann, who saw ten healthy children become infected with tuberculous ulcers of

the penis by the mouth of a tuberculous rabbi, in the course of ritual circumcision, seem to me to be instances of primary *tuberculous chancre*, comparable to the chancre of newly inoculated guinea-pigs, but not tuberculous ulcers like those of consumptives.

At the onset of a tuberculous ulcer, the lesion consists of one or several elevations of a dusky red color, which become lighter and finally evacuate their contents externally. The orifices become confluent and the ulcer extends rapidly on the surface, but slowly in depth.

I have previously described its typical features in its fully developed stage (page 288), a few of its most common clinical varieties, as well as the grounds on which the diagnosis rests; the reader is also referred to the description of atypical tuberculous ulcers elsewhere in this book (p. 291).

Treatment.—In a consumptive patient, it suffices to touch the ulcer with camphorated naphthol, lactic acid, peroxide water, methylene-blue, or with a mixture containing iodoform; the pain must be controlled with cocain; if dressings are applicable, iodoform or one of its substitutes, dermatol, airol, europhen, etc., should be employed.

If the patient does not appear to be tuberculous or only slightly so, the total removal of the lesion is indicated, or in default of this, scraping followed by cauterization with the thermocautery and appropriate dressings.

Tuberculous Gummas.—These are subcutaneous nodes or nodosities due to the Koch bacillus, which may be demonstrated in variable numbers. Their course tends toward softening and evacuation. They have been described elsewhere (p. 268).

They are encountered preferably, but not exclusively, in patients in a bad general condition, suffering from visceral tuberculosis or especially from bony or glandular tuberculosis. Children and youthful individuals are more particularly susceptible.

The *treatment* of tuberculous gummas varies according to their seat, their number and the general condition of the patient. When they can be extirpated surgically at an early date this is the most advisable procedure. Puncture followed by alterative injections rarely yields good results. When the gumma is open, it may be touched, after scraping if necessary, with iodin solution. Camphorated naphthol, iodoform-ether, guaïacol-oil, etc., followed by the application of dry or gomenol-oil dressings.

Tuberculosis Verrucosa.—The term tuberculosis verrucosa, in ordinary use since the very complete investigations of this dermatological type by Riehl and Paltauff in 1886, has replaced the terms verrucous scrofulide (Hardy, 1860), lupus verrucosus (MacCall Anderson, 1877) and sclerotic papillomatous lupus (Vidal and

Leloir, 1882), which more or less closely corresponded to it. The condition was very thoroughly discussed by H. Moutot, in his *Thèse de Lyon*, 1907 (Fig. 167).

Elsewhere in this book I have recorded the clinical description of the lesions and pointed out their usual site (page 247). The patches are sometimes multiple.

Tuberculosis verrucosa results in the great majority of the cases from an external inoculation.

It is observed in consumptives as the result of auto-inoculation of their sputum or dejecta; on the other hand, in nurses, butchers and veterinaries, who through their occupation come in contact with tuberculous individuals, human or animal. It may be derived from an auto-infection of the skin by a focus of bony, articular, or tendinous tuberculosis. Tuberculous lymphangitis, which usually

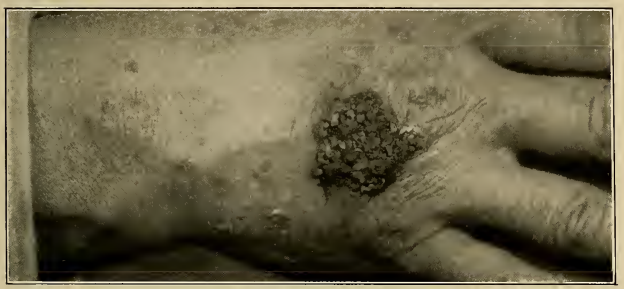


Fig. 167.—Tuberculosis cutis verrucosa.

assumes a gummous or fungoid type, sometimes presents a verrucous character. A few scanty Koch's bacilli may be found in the sections; guinea-pig inoculation is usually positive. Some authors believe that tuberculosis verrucosa is due especially to a bovine bacillus; according to others, a human bacillus is more commonly demonstrable.

The onset, which usually escapes notice, is in the form of a small, hard, horny nodule which frequently suppurates and becomes an abscess. The development of the lesions is centrifugal or serpiginous.

Lupus tubercles are never demonstrable in the developed stage, but I have noted their appearance in the subsequent cicatrix.

The duration is very protracted and indefinite. The patch may become partially cicatrized, but a complete spontaneous cure is rare. Tuberculosis verrucosa may lead to lymphatic propagation and visceral generalization; it accordingly constitutes a serious lesion.

Anatomical Tubercle — or *verruca necrogenica* — described by Laennec, is merely a particular instance of tuberculosis verrucosa. It is encountered among medical students, physicians, dissecting-room attendants and nurses; its more rapid course is undoubtedly explained by mixed infection. As early as the day following the local infection, through a puncture or contaminated erosion, a painful erythema makes its appearance, followed by the formation of an elevated dermic pustule. In spite of application of various topical agents, the elevation persists, becomes indurated and gradually extends through the confluence of neighboring small pustules. The lesion promptly assumes the appearance of a horny papilloma or a hard tubercle covered by a crust; it is surrounded by a purplish-red halo; it is painful on pressure. The mixed tuberculous and pyococcic infection may extend deeply, reaching the lymph channels and glands of the elbow and the axilla, causing suppurative adenitis and ultimately a visceral tuberculosis.

Treatment of Tuberculosis Verrucosa.—This must be early, rapid and energetic. The method of choice is surgical excision, when this is possible. In default of excision, excellent results are obtained by thorough scraping, under local anesthesia, followed by two or three radiotherapeutic sessions during the cicatrization. *X*-rays or radium employed alone are not to be recommended. Strong caustic agents, Vienna paste, Unna's white caustic paste, carbonic acid snow or superheated air, may also be employed in certain cases. The cicatrix must always be watched, as relapses are likely to occur.

Fungoid and Vegetative Tuberculosis.—In addition to tuberculosis verrucosa and lupus tumidus or vegetans, a less common form is observed, which is entitled to the name of *fungoid tuberculosis*, proposed by Riehl in 1894.

It presents the appearance, either of a very soft, irregular and lobulated red tumor, or of a flabby, distinctly outlined, mammillated and prominent patch. Its surface may be ulcerated, crusted or partly cicatrized.

It seems to result sometimes from an exogenous inoculation, in other cases from an auto-infection of the skin derived from a bony, articular or glandular focus. It is met especially on the limbs, but also on the trunk and even on the face. The differential diagnosis from mycosis fungoides, epithelioma, sarcoma, blastomycosis, sporotrichosis, etc., may present serious difficulties, but these can be overcome by the histological examination, bacteriological findings and animal inoculation.

The following may be considered as varieties of the above form: *Tuberculosis frambœsiformis* composed of large, soft and extensive papillomatous patches, occupying especially the perigenital regions; it was described by Doutrelepont, Wickham, Hallopeau, Jessner,

etc.; also the *tuberculosis fungosa serpiginosa* of Jadassohn; furthermore, certain *tuberculous lymphangitides* (fungoid lymphangitic tuberculosis) studied by Bazin, A. Fournier, Morel Lavallée, et al. In the last-named variety, which closely resembles the lymphangitic form of sporotrichosis (Fig. 180), a series of lesions are found arranged in succession along the course of lymphatic vessels coming from a tuberculous focus; these lesions may be fungoid or in other cases gummous or verrucous; sometimes a subcutaneous, nodular strand connects the various foci with each other. Hallopeau and Goupil described a *lymphangiectatic* variety, in which the tuberculous infiltration is complicated by lymphatic varicosities; it may lead to secondary elephantiasis.

The *treatment* of the fungoid tuberculoses must consist in scraping and cauterization. Radiotherapy may render valuable accessory services.

FIG. 168.—Lupus vulgaris (agminatus, tumidus, non-exedens) with central sclerosis and centrifugal extension.

Lupus Vulgaris.—Lupus—lupus vulgaris, tuberculosis luposa, Willan's lupus—owes its traditional name to the ulcerative, gnawing, devouring tendency frequently assumed by it. It represents the most common as well as the most polymorphous and most obstinate form of all cutaneous tuberculoses.

Lupus vulgaris is characterized by its eruptive lesion, the *lupus tubercle* or *lupoma* (page 256).

It is situated, as previously stated, on the face and neck in the great majority of the cases, especially at the nose or on the cheeks; more rarely on the limbs or on the trunk.

Varieties.—Lupus may assume extremely varied appearances. Such a large number of varieties have been described by authors

that one is often at a loss as to where to group a given case. Familiarity with at least the principal forms seems essential.

Three types of lupus are distinguished according to the *distribution* of the lupus tubercles:

Lupus disseminatus, in which isolated or sometimes very numerous lupomas are spread over one or more regions of the body; it usually occurs as a sequel of infectious diseases, especially measles; it closely resembles cutaneous sarcoid or lupoid.

Lupus agminatus is the most common form; the grouped tubercles become confluent in the center of the patch as a disk or plaque and are scattered in the circumference (Fig. 168).

Lupus diffusus or *confluens* consists of an often prominent, soft, nodular patch, of a purplish, yellowish or brownish-red color, with rounded, oval or irregular contours; the lupus tubercles are confluent and indistinguishable.

The degree of *prominence* of the lupus permits a differentiation between the following forms:

Lupus Planus.—This comprises numerous varieties, *macular, squamous, psoriatiform, erythematoid* and *colloid*, which are sufficiently characterized by their designation. Lupus planus is usually of the agminated type.

Lupus Tumidus or Lupus Elevatus.—This form, which is more common than the preceding, likewise presents varieties: *colloid, myxomatous*, etc.

Lupus Hypertrophicus.—An *angiomatous* variety has been described; a *papillomatous* variety which practically blends with tuberculosis verrucosa; and an *elephantiastic* variety which affects the limbs especially.

The *configuration* of a lupus patch is of interest only in so far as it is connected with the course of the affection. *Discoid, corymbiform, marginate, circinate, centrifugal, serpiginous, linear, annular* and other configurations have been described.

The developmental *tendency* of a given lupus is the most important of all its features. Rayer and Devergie already recognized the existence of two great classes from this point of view.

1. *Lupus non-exedens*, which does not ulcerate and manifests itself in type, form and configuration under one of the numerous varieties enumerated above. It grows slowly, continuously or intermittently.

It not infrequently becomes transformed into a cicatrix at its center; but a recurrent growth of the lupomas in the sclerotic tissue is far from uncommon. When spontaneous total cicatrization occurs, as in lupus planus and even in lupus tumidus, the condition is described as *scleroticus* or *resolutus*.

The *erythematoid* variety of lupus planus, also known as *ery-*

themato-tuberculous lupus of Vidal and Leloir, was considered by the last-named author as a combination of Willan's lupus with the lupus erythematodes of Cazenave. It gives rise to frequent errors in diagnosis. This is really a *superficial tuberculous lupus*, as properly recognized by Dubreuilh; the lupomata are small, subepidermic, sometimes confluent and not readily perceptible to the unaided eye; but they are distinctly revealed on biopsy. In this variety of lupus, which may invade the nose and the cheeks in the form of a butterfly, there is a pronounced tendency to spontaneous cure; it is of the resolutive type.

A lupus non-exedens may at any moment of its course become erosive, ulcerative and even mutilating; so that there is no essential difference between this class of lupus and the following:

2. *Lupus exedens*, or primarily ulcerative lupus, often takes a violent and rapid course; its exuberant proliferation, its active growth, its relatively rapid extension and the final partial disintegration undergone by it, lead to early destruction and mutilation. It assumes one of the following clinical forms:

Pustular lupus consists of soft elevations the size of a cherry pit, which have a tendency to become confluent and which in less than a month open like abscesses.

Ulcerative vegetative lupus is composed of soft fungoid infiltrations, producing for instance the appearance of a tomato, into which sharp instruments will cut as into butter; the ulceration which results from the breaking-down of the newformation, leads to fearful mutilations of the nose, the lips, the velum of the palate, the eyelids, etc. It is described as *lupus vorax* or lupus *phagedenicus* when its course is especially rapid.

Serpiginous tuberculo-ulcerative lupus, which may precede or follow a lupus non-exedens, is seen on the trunk and the limbs even more often than on the face and, like erythematoid lupus, preferably in aged individuals; it consists of a central cicatrix bordered by large pustular crusted, sometimes rupioid, lupus tubercles. It closely resembles the ulcerative-serpiginous syphilides and although its course is slower it may periodically progress very rapidly.

The varieties of lupus most commonly met with are, briefly: *lupus planus agminatus*, with a tendency toward central cicatrization; *lupus tumidus agminatus* in patches, level or protuberant; and tuberculo-crusted or tuberculo-ulcerative *lupus serpiginosus*.

It is extremely common for a lupus which has not been treated or even dressed to present itself covered with yellowish or brownish crusts; in such cases it is impossible at the first examination and before it has been cleansed to determine the variety to which it belongs. The formation of the crusts and of the erosions concealed by them is undoubtedly referable in part to a pyodermic

complication. Lupus which is thus disfigured is ordinarily called *impetiginous lupus* (Fig. 169).

All these morphological varieties really possess only a didactic and diagnostic value. Lupus vulgaris is *single* in character and may very readily assume a variable appearance and behavior at different points or different times.

Etiology. *Topography.* Lupus may appear at any age, but in more than one-half of the cases it manifests itself before the fifteenth and rarely after the thirtieth year. It is more common in the female sex and in Northern countries. A certain family predispo-

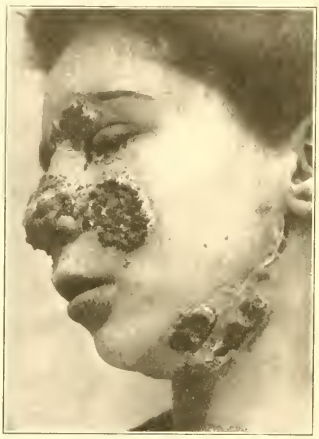


Fig. 169.—Lupus impetiginosus with multiple foci, showing the points of inoculation of the skin. The infection probably occurred through the nostrils and the nasal fossæ, reaching the nose and the cheek directly; attaining the internal angle of the eye through the lachrymal duct; the submaxillary region by way of the glands; a few of the latter were extirpated, as indicated by the keloid cicatrix under the ear; the other glands suppurated and opened on the skin.

sition seems to play a part; A. Ollivier mentions the case of a family of five children, four of whom had lupus. Very frequently an apparent tuberculosis is found in the ascendants or collaterals; however, as pointed out by Besnier, consumptives do not acquire lupus, whereas lupus patients very frequently develop pulmonary tuberculosis.

[All forms of lupus and especially the highly destructive forms are rare in America as contracted with their frequency in European countries. The high daily average of sunlight throughout North America may possibly play a role in this relative freedom, but on

the other hand, lupus is common in sunny Italy. It seems more probable in view of the fact that lupus is preëminently a disease of the poor, that the better nourishment of the poorer classes in America is the important factor.]

Lupus attacks principally, although not exclusively, individuals of a *scrofulous* habitus, with thick, soft flesh, a pale or florid complexion, swollen nose and lips, acro-asphyxia, frost-bite, chronic glandular enlargements and a readily vulnerable skin; in scrofulous women, the ear-lobes are often cut by the wearing of ear-rings. This classical scrofula is now known to correspond, not to a soil merely susceptible to the bacillus, but rather to an accomplished but attenuated and latent bacillary infection. Lupus accordingly seems to result from a local superinfection of autogenous or exogenous origin, in an individual in a state of allergy.

In the tissues of lupus, *bacilli* are very rare and dozens of sections must be examined before one is found. They have been assumed to be of a special strain or attenuated virulence, which would agree with the slow course of lupus. According to recent findings, the condition is more frequently due to the human than to the bovine bacillus; as to its virulence, the following statements can be made: Inoculation of lupus tissue, correctly grafted into the guinea-pig's peritoneum, gives rise, as a rule, to ordinary tuberculosis, transmissible to a series of animals; however, in my experience, the inoculation is unsuccessful in over one-third of the cases. Pautrier, who inoculated neighboring fragments of the same lupus into guinea-pigs, obtained, in the same series, sometimes positive and sometimes negative results. Whatever may be the explanation of these cases, it seems to me that in combination with the clinical data, they justify the interpretation of lupus as a relatively slightly virulent form of cutaneous tuberculosis and even as an intermediate pathological type between the true cutaneous tuberculoses and the tuberculides.

The *portal of entry* of the local infection which causes lupus is external or internal. The possibility of its resulting from an accidental inoculation of exogenous origin is attested by authentic cases in which a lupus has been seen to supervene in an apparently healthy person, following upon a wound, perforation of the ear lobules, vaccination, etc. Perhaps the ordinary impetigo of children may serve as an avenue of entrance. The frequency of lupus on the nose or in its vicinity is accounted for by the infection of the nasal fossæ through the dust inhaled in respiration or by contact with the fingers. As a matter of fact, four-fifths of lupus lesions are situated on the face (Fig. 169).

On the other hand, the origin of the superinfection may be autogenous and the inoculation of the skin take place from within out-

ward as in case of lupus following upon broken-down glands, of common occurrence in the neck; or upon tuberculous osteitis, as seen on the limbs. Lupus of the cheek may be due to a tuberculous lymphangitis derived from the nasal fossæ.

Cases in which the bacillus arrives from some internal focus by way of the vascular system are more numerous than is usually assumed. Disseminated lupus lesions are evidently produced by this means, favored especially by a temporary loss of body resistance under the influence of measles, grippe, pregnancy, etc.

On the genital organs, lupus is rare; in women, it has been confused with esthiomene. At the circumference of the anus, lupus is often combined with papillomatous tuberculosis or tuberculous ulcerations and results from an auto-inoculation with bacilli contained in the dejecta.

The palmar and plantar regions and, to a certain degree, the trunk but very particularly the scalp, are endowed with a relative immunity toward lupus.

Lupus of the mucous membranes is encountered in one-third of the cases, but must be looked for. I have pointed out that the initial focus is frequently nasal. Often soft elevations are observed on the anterior portion of the septum leading to perforation of the latter. From here, propagation takes place to the eyelids through the lachrymal passages, but the conjunctiva usually escapes; or to the palate and gums, through the anterior palatine foramen, and more frequently, to the velum of the palate and to the pharynx.

In the throat, in the mouth and the larynx, lupus usually assumes the appearance of a pinkish mammillated surface interspersed with ulcerations; on the gums, that of a crop of soft red fleshy proliferations; on the tongue it is very rare and almost invariably assumes a papillomatous form, although I have seen a lupus ulcer of the tongue.

In a general way, although lupus of the mucous membranes belongs to different varieties and although no lupomata of a barley-sugar appearance are here demonstrable, its course and the usual coëxistence of lupus of the skin serve to facilitate its diagnosis.

Course and Complications.—The long duration of lupus and its rebellious character are its most essential features. Lupus persisting for ten or twenty years is very common. Feulard reported a case of sixty-eight years' standing.

Its ulcerative forms, but also up to a certain point those with a tendency to cicatrization, lead to deformities and truly hideous *mutilations;* ectropion, complete destruction of the nose with stricture or even atresia of the nostrils, buccal atresia, vicious and keloid cicatrices and transformation of the extremities into misshapen stumps (Fig. 170) are not uncommon.

The *course* of lupus may be interrupted by congestive, edematous or suppurative exacerbations.

Recurrent lymphangitis seems to play a part in the elephantiastic form. *Erysipelas*, considered as favorable by some authors, appeared rather injurious in the majority of cases. Corresponding glandular enlargement or *adenopathy* is rather common although not constant.

Pulmonary and visceral tuberculosis is responsible for a large number of deaths among lupus patients, although it usually takes a slow and protracted course in these cases. Many patients affected with lupus otherwise enjoy excellent health. I have seen several who were married and had a healthy progeny.

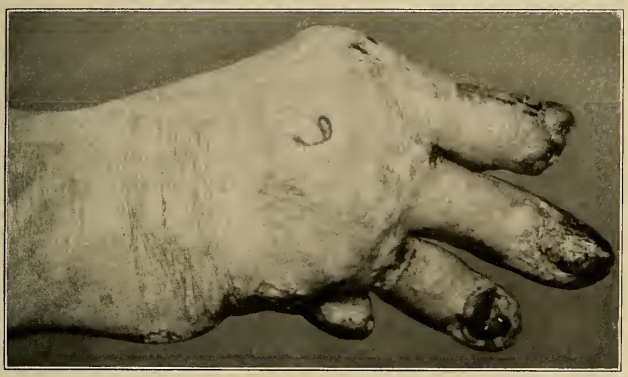


FIG. 170.—Lupus mutilans of the hand.

Epithelioma is a formidable complication of lupus, on account of its rapidly invasive course. Its frequency has been estimated at 4 per cent. of the cases. The onset of cancer is recognized by the changed appearance of an area of the lupus eruption, where a hard painful proliferation arises and promptly ulcerates or undergoes partial necrosis; biopsy is often required to confirm the diagnosis.

Tertiary or congenital *syphilis* has been charged with creating a predisposition to lupus; a possible association has even been suspected on the basis of the sometimes very favorable although incomplete effects of mercurial treatment in some cases of lupus.

Diagnosis.—The differentiation of lupus from tubercular or tuberculo-ulcerative tertiary syphilides is most frequently demanded. The course and the age of the lesions sometimes

furnish presumptive evidence; actually, leaving aside lupus exedens, it may be stated that lupus requires years to accomplish what syphilis will do in a few weeks or months. The softness, the yellow translucid color of the lupus nodule and its renewed growth in the cicatrix, are characteristic of lupus. However, certain syphilides are "lupoid" to such a degree, even on histological examination, that the question can be settled only by means of the Wassermann reaction and guinea-pig inoculation; the local tuberculin reaction also possesses a real value.

The color, the opacity, the elastic consistence and the anesthesia of the tubercles of leprosy are sufficiently characteristic to guard against error; all doubts will be removed by a general examination of the patient.

The confusion of lupus with eczema, impetigo or psoriasis is inexcusable, although it may assume the objective appearance of an impetiginous or psoriatiform eruption.

The lupoid sycosis of Brocq affects only the hairy regions, is distinctly cicatricial in the center and bordered by suppurating folliculitis.

The differentiation may prove very difficult between lupus disseminatus and the sarcoid of Boeck, which I have described under the name of disseminated miliary lupoid; histological examination and guinea-pig inoculation furnish the only reliable criteria. The boundaries between various forms of lupus and tuberculosis verrucosa, or tuberculosis fungoides and its varieties, are indefinite, so that well-informed dermatologists will group under one of these headings cases which by others are considered as lupus; at any rate, these dermatoses are of the same nature.

What I shall have to say further on in regard to lupus erythematodes, actinomycosis, the blastomycosis, the leishmanioses, etc., will probably suffice to permit the diagnosis of these affections.

The tuberculin test with Koch's tuberculin A, in subcutaneous injection, when it produces a positive local reaction to a minimum dose possesses real value not only for the recognition of lupus but for the determination of its clinically undemonstrable extent. The cuti-reaction of von Pirquet with crude tuberculin or Moro's procedure which consists in the comparative rubbing of a small lupus surface and a surface of healthy skin with tuberculin incorporated in lanolin, equal parts, may also be utilized.

Lupus of the mucous membranes may simulate very different affections; usually the coexistence of lupus of the skin is of great help. In primary cases, the differential diagnosis from focal syphilomata and from papillary epithelioma is extremely difficult; the differential features may be insufficient and biopsy as well as serodiagnosis become indispensable.

Treatment.—I shall refrain from entering into details on this inexhaustible topic, restricting myself to practical suggestions. It is evident that the seat, the extent of the lesions, the age, the social condition and the general health of the patient must influence the choice of the therapeutic method.

When a lupus is operable without mutilation, the preference must be accorded to surgical removal, with or without autoplastics; the tuberculin test should first be carried out in order to recognize its actual limits, following the advice of Neisser and Klingmüller. Extirpation should accordingly be the method of choice in lupus of the limbs, the trunk, the neck, unless it is too extensive.

Curettage, if necessary under anesthesia, may yield rapid but unreliable results, only mediocre from the cosmetic point of view; in all cases, this treatment should be combined with cauterization by the actual cautery or caustic agents, carefully watching the cicatrization, during which radiotherapy may usefully be employed.

Total destruction through cauterization (thermocautery, galvanocautery, superheated air) or through chemical caustics (such as trichloride of antimony, trichloracetic and hydrochloric acids, etc.) is open to the same objections.

Abroad, pyrogallol is freely used as a salve (from 5 to 20 per cent.) but it is very painful; the green salve of Unna (see Therapeutic Notes) seemed preferable to me.

Most frequently, graduated progressive methods together with physical agents should be resorted to, thereby stimulating or favoring the sclerotic process adopted by nature in cases of spontaneous cure.

Crossed linear scarifications would constitute an excellent curative procedure, in view of the qualities of the resulting cicatrix, but for the often considerable and even endless number of sessions required for this treatment. Nevertheless, scarifications are imperative in case of vegetative lupus or lupus vorax, which they control with remarkable rapidity; in lupus of the orifices of the face; and for the correction of lupus cicatrices with a persistent crop of tubercles.

Ignipuncture with the galvanocautery does not aim at the total destruction of the lupus, but like scarification tends to produce centers of cicatrization which, multiplied by later sessions, lead to confluence. This is a most valuable procedure for the reason that it is inexpensive, more rapid than scarification, easily applicable, particularly adapted to cases of lupus already modified by previous treatment and especially unrivalled in lupus of the mucous membranes.

As adjuvants of the preceding methods, or even sometimes exclusively, according to some authors, medicinal topical agents

have been recommended and may prove highly advantageous; I shall quote only potassium permanganate in strong solution or even in substance, pyrogallol, resorcin and especially mercurials in the form of plasters.

The introduction of new physical methods has partly transformed and certainly improved the traditional therapy of lupus.

The phototherapy of Finsen undoubtedly yields the best cosmetic results, moreover without pain; on the other hand, it is open to serious objections on account of the considerable number of sessions required (200 to 300 for a lupus of moderate extent), the very high cost of the apparatus and the sessions, as well as the failures, the proportion of which is not inconsiderable. The mercury vapor quartz lamp is more convenient and less expensive.

Radiotherapy and radiumtherapy have not entirely justified the expectations placed upon them. Their indication and their mode of employment in lupus are still a matter of controversy. However, the majority of authors are agreed in the repudiation of destructive doses which involve the risk of sometimes tardy but always formidable radiodermatitis. Injudicious repetition of the sessions involves the same dangers. In moderate doses, the radiations are very efficient in fungoid or papillomatous lupus and in superficially ulcerated lupus lesions. But the *x*-rays are especially to be recommended as adjuvants and I may say that at the present writing, galvanopuncture or periodical scarifications followed by radiotherapy in cautious dosage, seem to constitute the best treatment of lupus not amenable to surgical extirpation. Radium should be accorded the preference in cases of lupus of the mucous membranes or lesions difficult of access.

Internal treatment alone must never be relied on for the cure of a lupus, but there is often reason for and marked benefit to be expected from medication with calcium, arsenic, cod-liver oil and iodides; as well as from a sojourn in suitable watering-places by the seaside or on high mountains. This *climatotherapy* may be combined with *heliotherapy*, which is very beneficial when properly employed.

Among the various hypodermic injections which have been advocated, notably cacodylates, cantheridin, nuclein, thiosinamin, etc., in my opinion calomel injections are the only ones which can sometimes be recommended. When applied as in the treatment of syphilis, they are capable of producing remarkable improvement, but are found to be very unreliable; on the other hand, systematic injections, in gradually increased doses, of Koch's tuberculin A, which I have resumed after many other trials, have sometimes yielded remarkably rapid favorable results in my experience; great caution is required in their use.

The treatment of lupus needs insight, considerable patience and

skill in the variation and combination of the methods best adapted to the individual case as well as indefatigable perseverance in the management of partial relapses. A cure cannot be considered as practically certain until several years have elapsed after the disappearance of the visible lesions.

Tuberculides.—Since my suggestion in 1896 a number of apparently very dissimilar dermatoses have been grouped under this heading, whose relations with tuberculosis are proved by the following features.

The tuberculides themselves are frequently associated in the same individual and very often coexistent with glandular, bony, serous, visceral or cutaneous tuberculous manifestations; or the patients have a tuberculous history, or are found to be tuberculous later on.

The histological structure of the tuberculides is sometimes typically tubercular and identical with that of the most positive tuberculoses; in other cases it is non-tubercular, inflammatory and necrotic; but this is now known to be the case even in certain undoubtedly bacillary tuberculoses.

As a rule, no Koch's bacilli are found in the tuberculides; however, several authors report their discovery by means of the improved methods of Much-Gram, with antiformin, etc. Inoculation of their tissue into guinea-pigs is usually negative; but exceptions to this rule have occurred, diminishing the demonstrative value of the usual negative findings. Furthermore, it is noteworthy that Gougerot experimentally produced analogous lesions, which he regards as identical with tuberculides, by rubbing pure cultures of Koch's bacilli on the shaved skin of the guinea-pig.

The tuberculin test is often, but not invariably, positive in the tuberculides.

From the point of view of their clinical course, the tuberculides in general are distinguished from the cutaneous tuberculoses by their usual occurrence in a disseminated form, which is less common in the latter; and moreover by their marked tendency to spontaneous cure.

They appear in successive crops, without fever, without disturbance of the general condition; they often have a symmetrical distribution and a variable but rather prolonged duration. The various forms have a marked predilection for certain periods of life and for certain regions which are predominantly affected.

Clinical Forms.—As nearly all the forms of tuberculides were observed and described before their real nature was suspected, the names they bear are based on their morphological appearance, so that their nomenclature is a regular Joseph's coat. To bring

some order into this disorder, I recommend the following classification:

A. **Cutaneous Tuberculides.**—The seat of the lesions is dermo-epidermic in the following forms:

1. *Lichenoid tuberculides* (lichen scrofulosorum and lichen nitidus—these forms have already been sufficiently described (pp. 146, 138). I have seen also a few rare cases which simulated patches of lichen planus.

2. *Papulonecrotic tuberculides* (folliclis and acnitis of Barthélemy).

3. *Acneiform tuberculides* (acne cachecticorum of Hebra)—this is simply a variety of the preceding form.

4. *Lupoid tuberculides* (benign sarcoids or lupoids of Boeck (p. 259).

5. *Erythemato-atrophic tuberculides* (lupus erythematodes of Cazenave and certain atrophodermas).

6. *Erythematous tuberculides* (lupus erythematodes exanthematicus).

B. **Hypodermic Tuberculides.**—Three principal types have been described: the *erythema induratum* of Bazin, the *subcutaneous sarcoids* of Darier-Roussy and the *disseminated nodular sarcoids;* in all probability these are simply varieties of the same species (p. 272). Certain scrofulous gummas of attenuated virulence (p. 268) and a few atypical tuberculous ulcers (p. 291) are evidently related to this group.

C. **Doubtful Tuberculides.**—It is necessary to point out in this connection a certain number of affections whose relations with tuberculosis are emphasized by some authors but denied by others; these relations are problematical or perhaps indirect. Such conditions are:

Certain forms of *frost-bite* (pernio tuberculides) (p. 34).

Pityriasis rubra of Hebra-Jadassohn (erythrodermic tuberculide) (p. 118).

Certain forms of *parapsoriasis* (parapsoriatic tuberculides?) (p. 111).

Pityriasis rubra pilaris (erythrodermic perifollicular tuberculide?) (p. 399).

Perhaps certain cases of *eczema folliculorum* of Malcolm Morris and a few other authors (eczematoid tuberculides?) (p. 396).

Perhaps also *angiokeratoma* (angiokeratotic tuberculides) (p. 695).

This enumeration, lengthy as it is, does not exhaust the subject. As a matter of fact, only the relatively common and fairly well-established clinical types have so far been described under a special name. There exist more rare types as well as some with mixed features and transition forms as yet unclassifiable. I have seen

cases, for example, which might have been indifferently diagnosed as lupus pernio or folliclis or lupus erythematodes; all that could be stated was that the case was one of tuberculide. One would naturally hesitate to lay down exact rules for unique cases or for transition forms which are of exceptional occurrence.

Nature and Pathogenesis.—According to their mental proclivities, authors have conceived a different and even contradictory interpretation of the nature of these dermatoses. In the opinion of some, their relations with tuberculosis were extremely doubtful. Tuberculosis, they said, is a disease so widely distributed that its presence in a patient or his environment is irrelevant. The course of the tuberculides, the absence of really scientific proofs of their bacillary origin, justify neither their arbitrary grouping with the legitimate tuberculoses nor the assumption of their kinship with them.

Others, on the contrary, regarded the tuberculous nature of these affections as so unquestionable that it was actually proposed to abandon the provisional term of tuberculides and to incorporate this group among the cutaneous tuberculoses, of which they were supposed to represent still more attenuated types than lupus vulgaris, for example. The dissemination and customary symmetry of tuberculide eruptions were explained as due to the virus having travelled by the vascular route. With reference to the attenuated virulence of the lesions, various interpretations were suggested. The theory of Hallopeau, according to which the tuberculides are dependent, not on the bacilli themselves, but on their soluble toxins (*toxi-tuberculides*) met with many objections. The interpretation of Haury appeared much more probable; in his opinion the tuberculides result from embolisms of dead or attenuated bacilli, perhaps due to the struggle they had undergone against the defensive forces of the organism in a primary glandular or other infectious focus or in the blood current. These bacilli arrive in the skin while still capable of exciting a temporary local reaction and even small necrotic foci; but they succumb or disappear more or less rapidly—hence the spontaneous cure and non-inoculability of the lesions.

By applying to the interpretation of the tuberculides the prevalent view of tuberculous infection, their pathogenesis becomes considerably clearer. Tuberculosis is an almost universal infection; it heals very often to the point of becoming clinically latent; but it remains nevertheless capable of reviving under various influences and of giving rise to a discharge of bacilli; it creates a state of relative immunity which manifests itself by the phenomena of allergy. It thus becomes intelligible that the cutaneous manifestations resulting from bacillary embolisms may vary greatly in form and behavior, according to the degree of attenuation of the

bacilli, according to the number of imported bacilli, as well as according to the degree and kind of allergy enjoyed by the individual. But these incomplete data and speculations still require definite scientific confirmation.

Papulo-necrotic Tuberculides.—This clinical form was first described under the name of *lupus erythematodes disseminatus* (Boeck, 1880), then as *cicatricial folliculitis of the hairless parts* (Brocq), as *folliclis* and *acnitis* (Barthélemy) [as *hydradenitis destruens suppurativa* (Pollitzer)], as *hidrosadenitis suppurativa disseminata* (Dubreuilh); Kaposi called it *acne telangiectodes*.

It is not very rare and occurs especially in adolescents and young adults.

The eruption, consisting of a very variable number of lesions, appears in successive crops. It occupies preferably the hands and the fingers, the forearms (Fig. 171), the elbows, the circumference of the knees, the feet, the face, the ears and not infrequently also the trunk.

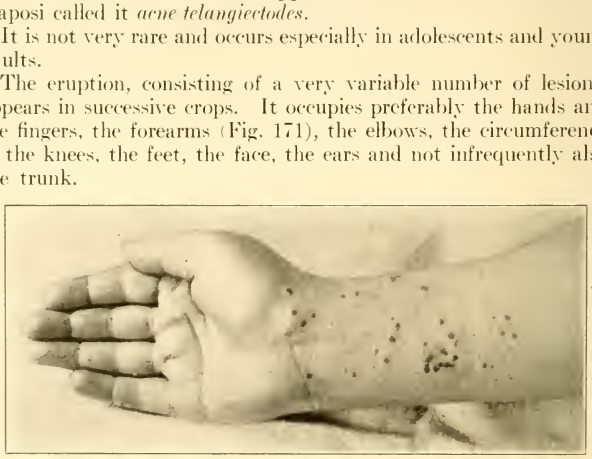


Fig. 171.—Papulonecrotic tuberculides. In this patient the eruption occurred on the four extremities.

The typical lesions begin as a very minute dull-red elevation, which on palpation reveals a hard painless nodule, the size of a pin-head, more or less deeply imbedded in the cutis; in about a week, this nodule becomes raised, its surface turns livid and purplish, while an epidermic vesico-pustule appears at its summit. When this is opened with a pin, only a very small amount of turbid serum escapes, and a sharp-bordered depression with a grayish floor appears in the cutis. Left unopened, the lesions become transformed into crusts which are not shed till after two to four weeks and leave distinct depressed cicatrices, often pigmented in their circumference and fairly characteristic. The crops following one another, the different stages are observed simultaneously. The total duration is several months or several years, with a tendency to seasonal recurrences.

With this principal type, which may be designated as *folliclis*, the following varieties are connected.

Acnitis *of Barthélemy* (Fig. 172) is seen in youthful or adult individuals, beginning and always predominating on the face, although sometimes scattered over the extensor surface of the limbs and the genital organs. The lesions consist of hard, miliary, distinctly circumscribed, isolated and indolent nodules, which originate in the depth of the cutis, may become absorbed, but more frequently suppurate and open on the surface, leaving a minute cicatrix behind. Each lesion lasts about one month, the eruption is continued over several months and is subject to recurrence.

FIG. 172.—Acnitis in a man aged twenty years.

Acne cachecticorum consists of slightly raised papulo-vesicular or superficial pustular miliary lesions, with a livid base, which appear in crops, without pruritus, in children or sometimes in adolescents or adults; they are situated principally on the limbs, to a small extent on the trunk and on the face and scalp. As a rule they are intermingled with lichen scrofulosorum or with typical papulo-necrotic tuberculides, of which they represent a superficial variety.

The name of *ecthyma terebrans scrofulosorum* may be applied to tuberculides related to the papulo-necrotic variety, but characterized by an eruption of lenticular or especially nummular, shallow and painless ulcerations which may coalesce in irregular patches; their course is slow, although occasionally acute.

With the exception of the acnitis type, which usually seems to be pure, the other varieties have a tendency to become combined with each other. They also become associated with small deep *nodosities*, analogous to scrofulous gummas of very small size.

Not to mention the coexistence of a local, glandular, etc., tuberculosis, which is common, the combination of papulo-necrotic tuberculides with acro-asphyxia, with pernio, with lichen scrofulosorum, with lupus erythematodes, etc., is far from unusual.

As their objective appearance is on the whole very polymorphous, when the various cases are taken into consideration, the differential *diagnosis* may have to be made from acne vulgaris, whose lesions, topography and course are entirely different; from some forms of folliculitis decalvans; and especially from papulo-crusted or follicular syphilides. Careful examination, however, will show that one is not dealing with the folliculoses, for the palmar and plantar regions may be involved; lesions have been met with on the buccal mucosa. Hidrosadenitis must likewise be excluded. In other cases the differentiation is not easy from frost-bite, or lupus erythematodes with small patches, or still other tuberculides.

The pathological anatomy will be discussed later.

The *treatment* must aim in the first place at correcting the general hygiene and nutrition. So-called anti-scrofulous medication with iodides, cod-liver oil and arsenic, is classical in these cases; calcium-salts remedies are indicated. I have obtained remarkable results from injections of calomel or soluble salts of mercury in cases with a negative Wassermann reaction. Still more serviceable are intravenous injections of arsenobenzol, combined with tuberculin injections in minimal and repeated doses. Locally, applications of ichthyol, methylene-blue, tincture of iodin or iodide powders, according as the lesions are ulcerated or not, seem to be useful. In obstinate cases, recourse may be had to climatotherapy, to heliotherapy and to cures at watering-places with saline, arsenic or sulphur springs.

Lupus Erythematodes.—Although clinically and histologically very different from lupus vulgaris or Willan's lupus, erythematous or Cazenave's lupus is likewise destructive and disfiguring; hence its name. There is not so much difference in its true nature; if lupus erythematodes is an erythemato-atrophic tuberculide, as there is good reason to believe.

Symptoms.—Lupus erythematodes consists of distinctly circumscribed red patches, covered with adherent scales; they are only slightly infiltrated and have a tendency to become atrophied at their center; their course is slow; they occupy with predilection the face, the ears, the scalp, the back of the hands and the fingers.

Each of these features deserves separate study.

The *redness* is constant, sometimes of a light pink or again of a carmine or livid shade. It results from a persistent erythematous inflammation and fades under vitropressure. Very frequently, however, the congestion is accompanied by the existence of a fine, reticular or stellate network of telangiectatic capillaries and sometimes by punctiform hemorrhages.

The *scaling* is very characteristic but varies greatly in degree. In typical cases, a very adherent, more or less continuous, hyperkeratosis is demonstrable, formed by fine, greasy, dirty-white or chalky lamellæ or stratified layers, which often look as if imbedded in a depression of the epidermis; rarely, the desquamation is psoriatiform. When this hyperkeratosis is slightly marked, it consists simply of a whitish stippling around the follicular orifices. The thick lamellar or chalky layers present small horny processes on their deep aspect, which penetrate into the follicles and corresponding depressions of the epidermis. This punctate cornification is one of the characteristic symptoms of lupus erythematodes.

The *infiltration* is very slightly marked and usually consists of a slight turgescence of the borders of the spot, the center being, on the contrary, depressed. Cardboard induration or discoid elevation of the patches is exceptional.

On the other hand, *cicatricial atrophy* is invariable and characteristic, although very variable in degree and depth. Unna, on this account, gave lupus erythematodes the name of *ulerythema centrifugum* (οὐλή = cicatrix). The cicatricial atrophy (p. 339) results from absorption, without ulceration, of part of the cutis and of the infiltration. When mild and superficial, it manifests itself as a delicate white sclerotic network, with grayish punctiform meshes, or as a thin and flexible, not very noticeable patch; it is usually depressed, pearly white and slightly indurated; the deep forms of lupus erythematodes leave a white sclerotic spot, without hairs and glands, sometimes squamous or pigmented.

The *tenderness* of lupus erythematodes spots on contact, pressure and especially scratching, is a sufficiently constant symptom to merit attention.

The *outlines* of the spots are sharp, rounded, oval or polycyclic; they are always bordered by a narrow erythematous margin, even when the central redness is concealed by the hyperkeratosis.

According to the relative importance of the dermic and the

epidermic lesions, a distinction is made between different clinical appearances which have received special names still sometimes employed: A case is described as *herpes cretaceus* of Devergie, when a massive surface hyperkeratosis is present; as *acneiform lupus*, when the hyperkeratosis is especially marked at the pilosebaceous orifices; as *seborrhea congestiva* (Hebra); or as *erythematofollicular lupus* (E. Besnier), when there are oily crusts provided with very apparent conical processes, penetrating into the follicular orifices; as *erythema centrifugum* of Biett, when congestion associated with a fine desquamation predominates. In *lupus exanthematicus* the lesions are still less marked.

It is noteworthy that in the majority of the cases the characters are not pronounced, or they are mixed so that their development is relied upon fully as much as their morphology for the description of the following varieties:

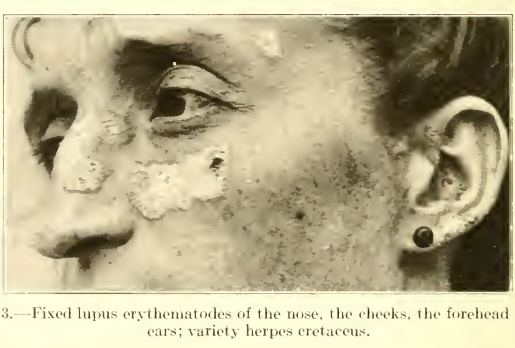


Fig. 173.—Fixed lupus erythematodes of the nose, the cheeks, the forehead and the ears; variety herpes cretaceus.

A. *Lupus erythematodes discoides*, or the *fixed* form, begins as one or several congestive, slightly infiltrated spots, promptly covered with adherent scales, spreading very slowly like a grease spot; their duration is counted by years. The hyperkeratosis and infiltration are usually very marked. The subsequent cicatrix is very atrophic; on the nose and ears it adheres to the cartilages which are themselves atrophic, hence rigidity, thinning and notable deformity of these parts. Recrudescence of the lesions in the cicatrix is not uncommon. This form is extremely obstinate.

Discoid or fixed lupus erythematodes (Fig. 173) preferably occupies the cheeks, the ridge of the nose, the temples, the ears, the forehead, the scalp, the neck and more rarely the back of the hands. Not infrequently it is arranged as a continuous patch symmetrically covering the nose and both cheeks, known as butter-

fly or bat-wing lupus (*vespertilio*). The spots, however, may also be irregularly scattered, sometimes there is only a single spot.

Localization on the buccal *mucosa* is generally considered as rare, although Th. Smith (1906) found it in 16 out of 56 cases. Most commonly it occupies the posterior aspect of the lower lip, where the extension of a red festooned or frayed surface from a lupus patch beginning on the free border of the lip may be observed. Cases of lupus erythematodes of the mucous surface of the cheeks or palate are not very numerous; lesions have been observed even on the tongue, in the form of a red or purplish spot with a parchment-like center and radiating leukoplasic margins, resembling buccal lichen planus. The diagnosis can be confirmed only on the basis of a coincident lupus erythematodes of the face, or through biopsy.

B. *Lupus Erythematodes Migrans* or *Erythema Centrifugum of Biett.*—Cases taking a relatively rapid course are connected by imperceptible transitions with stationary lupus erythematodes and their association is not uncommon.

A tendency to symmetry, to bat-wing form, to involvement of the ears and the hands, is much more marked in lupus migrans. The mucous membranes were never found to be involved.

The initial rose-colored spots grow in a few weeks, often presenting raised borders due to a soft infiltration and a central depression; they may become confluent in extensive surfaces.

Desquamation is moderate and may be compared to that of pityriasis or psoriasis or seborrhea.

The affection undergoes exacerbations, interrupted by long stationary stages. After healing, the cicatrix may be but slightly noticeable. The differential diagnosis from rosacea is sometimes difficult.

C. *Lupus Pernio.*—There is no agreement as to the identity of lupus pernio, lupus frost-bites or chilblain lupus of Hutchinson and its relations with lupus vulgaris on the one hand and with lupus erythematodes on the other. Lupus pernio presents itself in the form of a bluish-red edematous swelling with diffuse borders, occupying the nose, the malar regions, the ears, the back of the hands and the fingers and more rarely the toes; it develops under the same conditions as frost-bite and is not infrequently combined with acro-asphyxia, with papulo-necrotic tuberculides, with angiokeratoma, etc. Sometimes there is considerable analogy with Raynaud's disease. The lesions may become atrophied or ulcerated, giving rise to disfiguring scars. The affection lasts many years, with prolonged remissions which occur especially in the warm season.

When vitropressure or histological examination after biopsy

shows yellowish nodules analogous to lupus nodules in the spots or patches as is seen particularly on the nose, the condition would seem to be a *true lupus pernio*, representing a pathological type related to the sarcoids, for which the name of "benign lymphogranuloma" has been proposed (J. Schaumann).

Swollen erythematous patches, with an atrophic central depression, situated especially on the hands and fingers, may be said to be characteristic of *chilblain lupus*, which is probably related to lupus erythematodes. Both forms are entitled to classification under the heading of tuberculides.

D. *Lupus Erythematodes Exanthematicus.*—Under this name are described various rather rare *erythematous tuberculides*, characterized by lilac-red, finely scaly centrifugal spots becoming superficially confluent. They may spread in two or three weeks from the face and hands to the neck, the trunk and the limbs.

The *acute form*, described by Kaposi in 1872, has been observed either in young women already suffering from stationary lupus erythematodes of the face; or primarily. The primary variety was investigated in 1908 by G. Pernet, who collected 10 cases, 9 of which occurred in women. The course is rapid, febrile, with arthralgias, albuminuria and severe general disturbances; it almost invariably leads to death in a few weeks or months, through toxemia or through lesions of the respiratory apparatus, the kidneys or the meninges.

In the *subacute* form, the spots are disseminated and the general phenomena are absent, lengthy remissions occur and retrogression is possible.

The dermatosis designated as *erysipelas perstans faciei* by Kaposi, or as *erythema perstans* by Jadassohn, in which the red and edematous patches appear in a given region, preferably in the center of the face, possibly belongs to the same type.

Lupus erythematodes exanthematicus usually has no evident tendency toward atrophy.

Diagnosis.—Lupus erythematodes is distinguished: from rosacea, by the limitation of the red spots and by their scaly atrophic character; from psoriasis and psoriatiform eczematides, by its punctate adherent hyperkeratosis and by its tendency to atrophy; from senile keratosis, by the age of the patients, by its more pronounced redness and its non-verrucous surface.

The cicatrices of lupus erythematodes of the scalp differ from those of pseudofavus, favus and scleroderma in that they have followed upon a stage of severe reddening with hyperkeratosis. On the face or on the body, they must be distinguished from the idiopathic macular atrophies (p. 344); this is sometimes extremely difficult (Thibierge).

I repeat that the differential diagnosis from papulo-necrotic tuberculides, frost-bite and other tuberculides may be very puzzling, but is of minor importance.

Lupus erythematoides (p. 554) is characterized by lupomas which are sometimes difficult to make out without the aid of biopsy.

Etiology and Character.—Lupus erythematodes is observed very rarely in children before the eighteenth year or in aged individuals after sixty years; it is common from twenty-five to forty-five years, especially in the female sex and in cold climates. [In American dermatological practice it occurs once in about three hundred cases of skin diseases.] General circulatory disturbances, acro-asphyxia, a tendency to facial erythrosis and consequently the gastro-intestinal and utero-ovarian disturbances which are often primarily responsible for these conditions are thought to be predisposing factors.

The most interesting question, however, concerns the relations of lupus erythematodes with tuberculosis. It results from an international inquiry conducted by the *Annales de Dermatologie* (April, 1907), that only a little less than one-half the authors accept its tuberculous origin, which has been conceded in France since 1881. It has not been demonstrated, but the opposite theories, which claim a toxinic, nervous or microbic pathogenesis, are still less firmly grounded. Lupus erythematodes is interpreted by Brocq as a cutaneous reaction, which may result from a variety of causes, the most common cause being tuberculous infection. [This very nearly represents the opinion of Jadassohn also.] The following facts must be kept in mind: Many patients suffering from lupus erythematodes are tuberculous or very probably so; a few, however, seem actually free from tuberculosis. I have repeatedly seen patches of lupus erythematodes developing in connection with "scrofulous" lesions. The general tuberculin reaction is inconstant in these patients; the local reaction is absent in practically all of the cases. The histological lesions are inflammatory and not tubercular [in the anatomical sense]; giant cells are rare, no bacilli are found; these features are shared by several types of tuberculides.

However, although it must be admitted that the number of cases is very small, Gougerot as well as Br. Bloch and Fuchs succeeded in rendering guinea-pigs tuberculous through inoculation with tissue from lupus erythematodes.

Summarizing, it may be stated that lupus erythematodes is certainly not a cutaneous tuberculosis like lupus vulgaris; nevertheless, on account of its pathological relations, it may be grouped, although with some reservations, among the tuberculides.

Treatment.—Without being positively grave, lupus erythematodes is disfiguring and very obstinate; it is rare for patients suffering from the disease to reach an advanced age.

The local treatment must be governed by the clinical form. One must avoid attacking with caustic agents superficial spots which may disappear practically without a trace. In a general way, it is advisable to begin always by cautiously testing the patient's capacity of reaction, by means of astringent lotions, like lead water, or by applying weak ichthyol or resorcin pastes alternating with bland powders.

Gradually more energetic methods are employed and these must be varied in the course of the treatment. Painting with iodin, carbolic acid, arsenic; pyrogallol, resorcin, salicylic acid, oil of cade or mercurial plasters, etc., are usually valuable.

One of the best alterative agents is potash soap, which is applied during a time progressively lengthened from half an hour to eight or ten hours, to the point of producing an inflammatory reaction, which must then be controlled and the soap-treatment several times repeated.

In the superficial and actively extensive forms, high-frequency currents sometimes yield remarkable and rapid results.

In the stationary form, crossed linear scarifications or interstitial ignipuncture with the galvanocautery, more or less deep according to the case, constitute a classical treatment, the only objections to them being that they are rather painful and somewhat slow in action.

Phototherapy is rarely successful; radiotherapy or radium are preferable, but difficult to apply, requiring extreme care in this particular instance. [I have seen excellent results follow the use of the Kromayer lamp applied with pressure.]

On the whole, the therapy of lupus erythematodes is extremely disappointing. Patience and diplomacy are called for. It is important not to neglect hygienic measures and general treatment which is identical with that of an attenuated tuberculous infection. Long-continued administration of arsenic and especially of quinin in large doses, combined with iodin externally has been justly recommended.

Pathological Anatomy of the Cutaneous Tuberculoses and Tuberculides.—This group of diseases comprises an entire series of lesions, from the most obvious bacillary tubercles to atypical and relatively common reactions.

Tuberculosis.—There exists no tuberculous affection where better tubercles are found than in tuberculous ulcer (Fig. 101). It seems unnecessary to describe them here. They are arranged or scattered near the floor and the borders of the loss of substance, but are often only slightly evident near the surface of the ulcer itself. Koch's bacilli are abundantly present. Furthermore, there is a demonstrable inflammation of the connective and muscular tissues, as

well as vascular lesions; their degree and their extent are variable, in different cases.

The yellow granules of Trelat are due sometimes to intrapapillary or subpapillary agminated tubercles, undergoing a caseous degeneration; in other cases to small superficial abscesses due to secondary infections.

A tuberculous gumma in its formative stage consists of a mass of tubercles with typical nodules, in course of caseation. Bacilli are rarely present in considerable number. Sometimes the new-formation is encysted in a fibrous sheath, perhaps resulting from the dilatation of a lymph-vessel cavity; more frequently its boundaries are diffuse. In the stage of evacuation of the gumma, the tubercles are hidden in an abundant inflammatory tissue where they are not so readily demonstrable.

In tuberculosis verrucosa, lupus scleroticus and anatomical tubercle, there are well-marked hyperkeratosis and hypergranulosis, hypertrophied interpapillary proliferations, elongated but very irregular and uneven papillæ. The appearance at first sight is that of an ordinary papilloma, sometimes of a verrucous nevus. But the papillary body and the cutis are infiltrated with abundant round cells, sometimes collected in miliary abscesses. Moreover, scanty but very typical tubercles separated by sclerotic tissue may be seen here and there in the deeper portions of the cutis. Bacilli are present in variable number; the result of guinea-pig inoculation is always positive.

In fungoid tuberculosis, the tubercles and the bacilli are very thinly scattered in ordinary infiltration tissue.

The pathological anatomy of lupus vulgaris is somewhat different in different cases. The most characteristic appearance has been described elsewhere in this book (Fig. 89). The collections of tubercles may be rich in giant cells or without these and composed solely of epithelioid cells; sometimes the latter are absent and nothing is demonstrable but a diffuse infiltration of lymphoid and plasma cells, interspersed with enormous giant cells; the last-named structure belongs especially to ulcerative and exuberant lupus.

The lupus tissue in all cases is distinctly circumscribed; the connective tissue and elastic fibers stop at its circumference; this fact explains the softness, the translucidity and the very evident clinical boundary of lupomata.

The lupus infiltration is situated at a very variable depth according to the clinical form; under the epidermis, in the corium, or as far as the hypoderm. In the last named layer or perhaps at a certain distance from the apparent nodule, it is not uncommon to meet with tuberculous infiltrations following the lymphatic or venous routes, or with aberrant islands, accounting for the very frequent recrudescences after a destruction supposed to be complete.

The connective tissue of the vicinity is often edematous and interspersed with lymphoid cells, sometimes frankly suppurating in cases of lupus with a rapid course. The question has arisen: is this inflammatory reaction due to a special virulence of the bacillus or to secondary infections? In other cases, there is a fibrous reaction with a tendency to encapsulation of the newformation, more particularly in the forms capable of undergoing absorption. Lupus cicatrices often enclose latent nodules.

The epidermis is sometimes passive, distended and atrophic, whereas in other cases it proliferates, vegetates and may grow so exuberantly that certain vegetating or papillomatous cases of lupus or framboesiform tuberculosis are suggestive of lupus-epithelioma. In proliferating lupus of an ulcerative appearance, the epidermis is often found to be reduced to its deep layers.

Lupus is so poor in bacilli that their demonstration discourages the majority of histologists and very few have pursued it to the end. From forty to sixty slides often must be studied before a bacillus is encountered. The human bacillus is responsible for most of the cases.

Inoculation into guinea-pigs, which react readily, necessitates the injection of at least 50 cgm. of lupus tissue, to be convincing; even under these conditions, it fails in one-third of the cases. The virulence of the bacillus is usually moderate and exceptionally attenuated. The tuberculin test, which is always positive requires doses of $\frac{1}{10}$ of a milligram to 1.5 mgr.

On the whole, lupus vulgaris is certainly a cutaneous tuberculosis, although in numerous respects it is related to the tuberculides.

Tuberculides.—The histological lesions of the tuberculides are not uniform in character. Sometimes they have a tubercular structure, like the most positive bacillary tuberculosis, representing what I have named the tubercular type, or Type A; in other cases, they are of ordinary inflammatory character, representing my non-tubercular type B (also named atypical cutaneous tuberculosis by Pautrier, non-tubercular inflammatory tuberculosis, by Gougerot and numerous authors). I was led formerly to believe that the different tuberculides followed one or the other type of lesion, but have since recognized that the two aspects may coexist or succeed one another in the same clinical form.

At any rate, it is usual to find typical tubercles in lichen scrofulosorum, where they are situated at the level of the papillary body; they are also met with in the hypodermic sarcoids and in erythema induratum.

In the cutaneous sarcoids of Boeck, the infiltration as in some cases of lupus vulgaris and especially in lupus pernio, is composed for the most part of circumscribed collections of epithelioid cells.

In the papulonecrotic tuberculides, the initial lesion is a small necrotic focus of the connective tissue which promptly becomes surrounded by a reactive inflammatory zone, whose lymphoid and embryonic cells are preferably arranged around the vessels. These more or less deeply situated foci, very superficial in acne cachecticorum tend to rise and come to lie under the epidermis which is raised by a little serum, finally drying up in small crusts. In acnitis (Darier) and at the terminal stage of the papulonecrotic tuberculides (Brissy), very distinct tubercles may be encountered.

The essential lesions of lupus erythematodes consist of the following: in the cutis, a diffuse and especially perivascular cellular infiltration, composed of small connective-tissue cells and lymphocytes; plasmocytes and polynuclears are rare. Audry and Leredde found giant cells, which are of exceptional occurrence and usually associated with a disintegrated hair-follicle. Some bloodvessels are obliterated while others are dilated; small hemorrhagic points, lymphatic dilatations and often edema in the papillary body occur.

The epidermis is in a state of partial horny atrophy, meaning that its Malpighian layer after at first having been thickened, is now atrophied and locally reduced to one or two rows of deformed cells; its stratum granulosum is missing at numerous points, the horny layer on the contrary is thickened, stratified and penetrates in cones into the sebaceous and sudoriparous pores and into the interpapillary buds; the last named fact accounting for the clinical sign of "punctate keratosis."

The disappearance of the elastic and connective-tissue fibers where the infiltration predominates, indicates the mechanism to which the final atrophy is due. The hairs fall out soon. The originally enlarged glands finally disappear.

Briefly, the condition is an endoperivasculitis with a tendency to atrophy and to hyperkeratotic atrophy of the epidermis.

Koch's bacilli have been discovered in lichen scrofulosorum (Jacobi and Wolff) and in erythema induratum; with the aid of modern methods, with antiformin or by the Gram-Much procedure, positive results although open to controversy have, moreover, been obtained in nearly all the tuberculides, including lupus erythematodes.

In exceptional instances, guinea-pigs have been successfully rendered tuberculous through lichen scrofulosorum (Jacobi, Wolff, Pellizari, Haushalter, Lefebvre), through erythema induratum (Thibierge and Ravaut, C. Fox, Eyre, Carle), and through lupus erythematodes (Gougerot, Br. Block). Inoculation into monkeys is not more apt to be successful.

The tuberculin test yields inconstant and often not very distinct results, although Jadassohn obtained a positive local and general

reaction in 14 of 16 cases of lichen scrofulosorum; it is also often obtained in erythema induratum. I was equally successful in the three cases of subcutaneous sarcoids which were examined from this point of view.

LEPROSY.

Leprosy (lepra of the Arabs, elephantiasis of the Greeks, spedalsked of the Norwegians, la lèpre of the French, Aussatz of the Germans) is a chronic infectious disease, which develops with periodic exacerbations and is caused by a special microbe, the Hansen bacillus. Numerous and varied cutaneous manifestations occupy a prominent place in the symptomatology of the disease.

Leprosy seems to have existed from the earliest times among the peoples whose history has come down to us, notably in India, Egypt, Greece (alphos), China, etc. But it was confused with numerous contagious affections, so that the *zaarath* of the Bible has nothing in common with genuine leprosy (W. Dubreuilh and Bargues). In the middle ages, after the Crusades, it became widely distributed in Europe; numerous leper asylums were erected (over 2000 in France) which in all probability erroneously harbored more than one case of tuberculosis or syphilis.

[Originating in the Orient, leprosy was spread over Europe by the Roman armies and colonizers. There are ample records of its presence in France and England long before the Crusades but that great movement of men to and fro across Europe served to increase the number of cases enormously. In the fourteenth and fifteenth centuries the disease was common throughout the British Isles. Thereafter the epidemic gradually faded and the last autochthonous leper died in the Hebrides about a hundred years ago. The disease was carried to the West Indies, Mexico, Central and South America by the early Spanish settlers and by negro slaves from Africa. It reached the shores of the United States in the same way and more directly from the West Indies. In the Philippines with their large Spanish and Chinese population leprosy has been endemic for several centuries. In the Sandwich Islands it was introduced about the middle of the last century.]

At the present day this disease is practically extinct in our regions. Autochthonous cases are rare in France, even in Brittany and Provence; Great Britain, Belgium, Switzerland, Germany, Austria, as well as the United States of America, are free from it or have only a few trifling local endemic foci. Lepers are more common in Norway, Italy, Spain and Algeria. In the Balkan Peninsula, in Southern Russia, in the Baltic Provinces and in Iceland, leprosy is fairly common.

Among the most gravely infected countries, I shall mention

Hindustan, Persia, China, Indo-China, Japan, Polynesia and notably New Caledonia and the Sandwich Islands; Latin America [including some of the West Indies] may be considered as an important focus; certain parts of Africa are also more or less severely affected.

[In the United States the largest focus at present is in Louisiana where it has existed since the first settlement. In the middle of the last century a considerable number of cases were imported into the Northwestern States by Scandinavian immigrants, but there has been no increase in these regions; on the Pacific Coast a small focus is due to importation from the Sandwich Islands and the Orient. There are a few sporadic cases in Texas, Florida and South Carolina. In the larger cities, cases are found among the emigrants from Russia, Italy, the West Indies, South America and China.]

Owing to the facility and increasing frequency of communication, there are numerous opportunities for the importation of lepers into seaports and large cities all over the world.

The authors to whom we are indebted for most of our knowledge on the subject of leprosy are: Danielssen and Boeck, Sr. (1846), Virchow, Hansen, Neisser, Besnier, Leloir, Unna, Zambaco, Ehlers, Jeanselme, etc.

Etiology.—Leprosy is a disease of mankind and respects no race, no age, no social station, nor any climate or latitude.

Leprosy is caused by the bacillus which was discovered by Hansen in 1871 and was more accurately studied and stained by Neisser. It resembles the Koch bacillus, but is shorter, more rigid, less regular in form and much more abundant in the lesions; it is acid-fast and stains by the Ziehl method as well as by the methods of Gram, Weigert and Much.

It is not certain that real cultures of the Hansen bacillus have so far been obtained; of the positive results reported by numerous authors on more or less complicated culture-media, none have proved universally convincing.

Inoculation of leprous tissue into all sorts of animals is usually a failure; but Ch. Nicolle, in 1905, apparently succeeded in infecting a Macacus sinicus, in which nodules made their appearance on the sixty-second day.

The disease known as "rat leprosy," discovered by Stefansky in Odessa in 1903, which is distributed over the entire world, presents in certain respects, notably in the characteristics of its pathogenic agent, analogies with human leprosy, although it also differs from it very essentially. E. Marchoux, who recently investigated its modes of propagation in view of possible deductions applicable to human leprosy, found that it is transmitted especially through

bites or contact with an erosion, with flies as an accessory factor and perhaps sarcoptes and demodex, but not stinging insects.

Human leprosy has long been considered as hereditary, although this interpretation still has its advocates, it is not proved by indisputable facts; on the contrary, the children of lepers are known to remain free from the disease, provided they are removed at birth from the infectious focus. Few births occur among leprous families.

The contagiousness of leprosy, on the contrary, is unquestionable, but its mechanism is not known and it seems to be subject to certain special conditions. Thus lepers who have been imported into countries free from the disease, for example Paris [or New York], where more than 150 lepers permanently reside, do not establish a local epidemic focus. Nevertheless Frenchmen often contract the disease in countries where leprosy prevails. This fact seems to support the theory of an intermediate host or parasitic carrier of the contagium, such as a mosquito which does not exist everywhere.

Exceptional but demonstrative cases of transmission are, however, known to have occurred in our countries, like that of Hawtrey Benson's Irishman who was infected after his leprous brother's return from India, whose bed he had shared; or that of Veyrierès' patient who contracted the disease in Nice through her husband.

In this connection may be mentioned the extremely rapid propagation of leprosy in the Hawaiian Islands and in New Caledonia, for instance, where a considerable portion of the population was infected in the course of ten or fifteen years.

Poverty, dirt, promiscuity and prolonged contact are emphasized as favoring factors of contagion, which is supposed to occur indirectly, through the intermediation of clothing or ordinary objects. On the other hand, the presence of ulcerated tubercles, the discharge of secretions containing large numbers of bacilli, as is often true for the nasal mucus, the saliva and the genital secretions, serve to render the cases of "open leprosy" more dangerous. However, the portal of entry in the contaminated person is unknown; the nasal fossæ in particular, perhaps the digestive tube, the skin, especially the skin of the feet in bare-foot populations, have been considered as possible atria.

Direct inoculation from man to man, accidental or experimental, is often ineffectual and demands great caution when made in countries where leprosy prevails. But the experiment performed by Arning, in the Hawaiian Islands, on the convict Keanu, in whom leprosy made its first appearance in the vicinity of the inoculated point, leaves little room for doubt.

The *incubation* of leprosy has a duration certainly very variable and often very prolonged.

Its medium duration fluctuates between three and five years;

sometimes it seems reduced to a few months; often it is prolonged up to ten years and extreme cases have been quoted of fourteen years (Landouzy) and of thirty-two years (?) (Hallopeau). It will be readily understood that the incubation period cannot always be accurately established.

Symptomatology.—*Period of Invasion.*—The first manifestations frequently consist of general disturbances of a rather ordinary character.

Depression, weakness, anemia and somnolence, are practically constant, as well as rheumatoid pains, arthralgias, backache and neuralgias; digestive disturbances are also noted (anorexia, dyspepsia, coated tongue, transitory diarrheas) headache and vertigo.

Fever is inconstant and often inconsiderable; but it may assume the form of intermittent attacks with temperatures of 104° and 105°, suggestive of malaria.

Absence of sweating, or on the contrary attacks of perspiration, severe itching or tingling sensations and a persistent feeling of cold, have also been observed.

Signs of a certain value are furnished by a dull coloration of the skin of the extremities, sometimes with local asphyxia or syncope; also by obstinate coryza or abnormal dryness of the nasal fossæ with recurrent epistaxis occurring without an obvious cause. All these symptoms, the nature of which is often misunderstood, persist during months and years, extending into the subsequent periods.

According to the tendency of the lesions to manifest themselves especially on the integument or on the nervous system, a distinction is made between tubercular leprosy [*lepra tuberosum*] and nervous leprosy [*lepra nervorum*].

Macular Period.—No matter what is to be the ultimate form of the disease, but especially in the tubercular form, an established leprosy generally manifests itself by spots or *leprides*, varying greatly in color, dimensions, abundance and duration, developing in irregular crops.

They occupy the face, the extremities, the extensor side of the limbs, especially the buttocks and the back. They may assume the appearance of a polymorphous, papular or nodular erythema, with general symptoms, taking a sluggish course. Most commonly they appear in the form of erythemato-pigmentary spots, of variable size (Fig. 174), sometimes simply congestive, or purely achromic or hyperchromic. Although not infrequently pruritic or painful at the onset, they are nearly always characterized by their hypoesthesia or their *anesthesia*, which is usually of the thermo-analgesic type. They are diffuse or circumscribed and frequently arranged in circles, rings or serpiginous lines. After the first appearance they

are often ephemeral and may vanish without leaving a trace; later on they become persistent.

The erythematous spots may cover large areas, simulating erysipelas or polymorphous erythema; as a rule, they soon become more or less purplish or bronzed or copper-colored, or less colored than the background in the colored races, and are covered with fine scales; transitory at first, they persist after a recrudescence, spreading like a drop of oil and becoming decolorized at the center. The tubercles usually develop upon these spots.

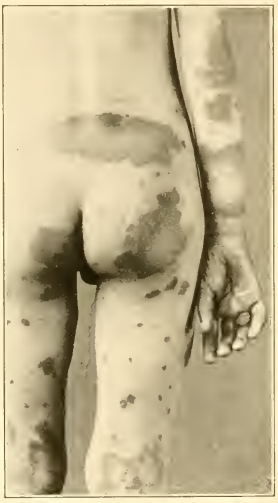


FIG. 174.—Erythemato-pigmented leprides, in a boy aged eleven years, son of a French official in Cayenne.

The pure pigmentary or achromic spots, belonging more particularly to the nervous form, present all the above-mentioned shapes and an entire color-scale, from black at one end to pure white at the other. They are often leuko-melanodermic, the center being depigmented and the circumference hyperchromic (*morphea leprosa*). They may simulate vitiligo (*vitiligo gravior*), chloasma, pigmentary syphilides, pityriasis versicolor, etc., but are characterized by their anesthesia. In colored individuals, leprous achromia produces a variety of "piebald negroes."

The combination with erythematous spots is fairly common and gives rise to peculiar checkered designs.

Following the macular period, or in place of it, *pemphigus leprosus* may occur, being much more commonly observed in the nervous form. It consists of voluminous but not very numerous bullæ, preferably affecting the back of the hands and feet, the elbows and the knees and often the surface of the achromic spots. Evacuation of their clear contents leaves a red or inflamed excoriation which on healing leaves a nacreous anesthetic cicatrix, with pigmented borders, possessing great diagnostic value.

The crops of bullæ recur in the course of the trophoneurotic stage.

Tubercular leprosy [*lepra tuberosum*] the tubercular or nodular form of leprosy—or *lèpre systematisée tégumentaire* of Leloir—is characterized by leprous tubercles or lepromata.

A tubercle which remained solitary for several months, has in rare instances been the first manifestation of the disease, as observed by Leloir, Marcano and Wurtz, and Gougerot; by analogy, this has been described as leprous chancre.

The tubercles are sometimes derived from the slow and partial transformation of erythemato-pigmented leprides; in other cases they develop in crops with abundant lesions, sometimes with febrile or afebrile general disturbances.

Their vaguely symmetrical localization is approximately that of the spots, with a predilection for the face and ears and possible extension to the mucous membranes.

Hypodermic lepromata are also observed, perceptible to the touch as circumscribed nodosities or infiltrated and nodular patches (Chapter XIV).

The leprous tubercles (Fig. 175) and the cutaneous leprous infiltrations have been described elsewhere in this book (p. 258), and I have pointed out their possible termination in leprous morphea and in leprous ulcers.

There still remains to be traced the picture of the topographical *distribution* of lepromata. Not infrequently they lend a characteristic appearance to the invaded regions. In the face, this aspect is described as *facies leonina* or *leontiasis leprosa;* the forehead is puckered, traversed by deep wrinkles; the region of the eyebrows is thickened and hairless; the nose is deformed, thickened and enlarged; the cheeks, the lips and the chin are lobulated and the beard is reduced to a few thinly scattered hairs. All these surfaces are of a red, brownish or grayish color. In very pronounced cases, the disfigurement is such that the race, age and sex of the patient are no longer recognizable.

The ears of lepers are typical, with an enlarged auricle studded with tubercles or seamed with cicatrices, a thick, hanging and flabby lobule in which numerous "birdshot" granules are demonstrable on palpation.

The scalp almost invariably escapes and the luxuriant hair on the head of lepers contrasts with their hairless face and the loss of the body-hairs.

On the extremities, the elbows, the knees and the prominent portions, including the fingers and toes, are deformed by brownish or purplish tubercles. The integument as a whole has a dusky, tawny, earthy, in places cyanotic color, a flabby, withered consistence and a peculiar dryness. The nails are dry, brittle and may fall out. In the lower limbs, a pachydermatous condition justifying the name of elephantiasis of the Greeks is not uncommon.

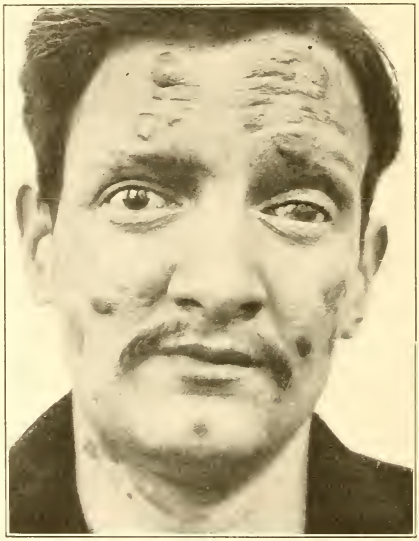


FIG. 175.—Leprous tubercles of the face. Note the alopecia of the eye-brows and interstitial keratitis, with iritis of the left eye.

Lymph glands are usually enlarged early and may attain an enormous size, without, however, undergoing suppuration.

Localization on the mucous membranes and on the special sense organs is very common.

In the nasal fossæ, the obstinate bacilliferous coryza and epistaxis of the onset are followed by lepromata or ulcers of the septum, leading to perforation as in syphilis, with breaking down of the nose.

The mouth may be the seat of lepromata or cicatrices affecting

the palate, the velum, the uvula, the pharynx and the back of the tongue.

The larynx is often attacked at an early date, hence hoarseness of the voice, aphonia, dyspnea after exertion and later on attacks of suffocation.

The eye is affected with deplorable frequency, especially in its anterior hemisphere. Leprosy here very rapidly gives rise to tubercles on the conjunctiva, interstitial keratitis with superficial pannus, episcleritis, iritis; the bacillary infiltration of the iris is sometimes manifested by nodules, almost invariably by exudates which impair vision. The ciliary body is more frequently invaded than the choroid and the retina. Attacks of glaucoma, secondary cataract and atrophy of the eye-ball, may hasten the loss of the eye.

Visceral leprosy is here only mentioned by name; it affects the lungs, where the differential diagnosis from tuberculosis which sometimes complicates it, is not easy but possible by bacteriological methods; the digestive tract, the liver, the spleen, the circulatory apparatus, etc., may also be affected.

Leprosy of the genital apparatus is common, especially in man. Orchitis leprosa is observed in at least one-third or one-fourth of the cases; it may be acute at the onset; as a rule it is an insidious bilateral orchi-epididymitis, of which the patient is not aware; the organ is smooth or nodular, but hard and undergoes atrophy. The lesions lead to early sterility, without loss of libido and later to impotence. Not many data are available on leprosy of the female genital apparatus; its different parts, notably the ovaries, may be damaged and undergo sclerotic atrophic changes; according to Babes, about 70 per cent. of female lepers are sterile.

The course of tubercular leprosy is very variable; it is rarely acute and fatal in a few months, but usually chronic, lasting ten or twenty years and longer. The attacks are interrupted by prolonged remissions, with retrogression of all symptoms, simulating a cure. These remissions are the rule in our countries. When the course is progressive, the ulcerations, the suppuration, the fever and diarrhea lead to marasmus and death, which often occurs as the result of severe local disturbance or a superadded complication.

Lepra Nervorum.—The second ordinary type of established leprosy is the *generalized nervous form* or *lepra maculo-anesthetica* or *trophoneurotica*.

It likewise begins almost invariably with macules; it is not certain that these may be entirely absent. Frequently they are large and perfectly symmetrical; disturbances of pigmentation are marked. The macular stage is sometimes indefinitely prolonged, so that a form of so-called macular leprosy [*lepra maculosa*] has been described.

In other cases, the onset of the disease is marked by pemphigus

leprosus which occurs in attacks sometimes leading to sloughs and frightful mutilations; some authors have accordingly recognized a macular and bullous form of the disease, known as *Lazarine leprosy*.

As a rule, nerve leprosy is characterized by tumefaction of certain nerves, by anesthesia and by trophic disturbances of the skin, the muscles and the bony framework.

The nerves accessible to palpation, notably the ulnar nerve above the bend of the elbow, the external popliteal, or the superficial nerves of the cervical plexus or the forearm, are cylindrically thickened or more often the seat of spindle-shaped swellings or beaded nodosities.

Although painful at first, they soon become insensitive and at the same time cutaneous anesthesia is developed.

Neuritis manifests itself by neuralgic pains, sometimes intolerable, circumscribed areas of pain, pruritus unrelieved by scratching, sensations of numbness, "dead fingers," local cyanosis, sudoral disturbances, etc.

The anesthesia, which possesses great diagnostic value, was carefully studied by Jeanselme. It particularly affects the extremities symmetrically, first the lower limbs, progressing from the periphery to the center. Band-like at the onset, occupying for instance on the upper extremities the little finger and the ulnar border as far as the axilla, at the lower limb the big toe and the internal border of the foot and leg, it subsequently assumes a segmentary type. It is variably distributed; outside of the territory where it is established, there exists a movable anesthetic zone, imperfectly bounded by a transitional area; at its origin it is superficial, but shows a tendency to increase in depth. The anesthesia is dissociated; the temperature sense is lost first, next, sensibility to pain disappears and much later the tactile and pressure sensations. Sensory perversions and retarded sensation are not uncommon. Finally the loss of sensation becomes total, at least at the extremities; deep burns may occur, the patient remaining unconscious of them.

Muscular atrophy affects especially the face and the extremities. In the face, the orbicularis palpebrarum is first attacked, as a rule, causing inability to close the eyes and its sequelæ.

The forehead, the cheeks, the circumference of the mouth are attacked in their turn. The emaciation of the face, the pale and wan complexion, the fixity of the features with lagophthalmos, lend the face a strange appearance, known as "facies Antonina."

On the hands, the muscular atrophy affecting the thenar and hypothenar eminences as well as the interossei, leads to deformity in the form of *ulnar claw*, or of boat-shaped hollows on the back of the hand, or to one of the types of chronic rheumatism. It later involves the forearm and especially the extensors and sometimes the upper arm. There is no paralysis but only a diminution in strength pro-

portionate to the atrophy. On the feet, the plantar muscles are the first to be attacked, but the atrophy of the anterior tibial and the extensors of the toes attract more attention, causing a varus equinus position and a halting gait.

The bony and articular lesions which are characteristic of *lepra mutilans* are derived either from penetrating ulcers, or from *malum perforans*, which is frequent and is distinguished by its depth and total anesthesia, or from panaritium terminating in necrosis, as in Morvan's syndrome; or from dry gangrenes; and often also from bone absorption without external lesion. The foot and the hand may be reduced to irregular stumps which have been compared to elephants' feet (elephantiasis of the Greeks) or to the paws of seals.

Nerve leprosy takes a slower course and has an even longer duration than tubercular leprosy; it is not uncommon for it to exceed twenty years. In the advanced stages, the condition of the patients is extremely distressing; they are emaciated, mutilated, blinded, paralyzed, suffer from intolerable neuralgias, persistent cold, unquenchable thirst, often from infected ulcers and are sunk in deep apathy, melancholia and marasmus. A toxemic psychosis of lepers has recently been described. Death supervenes through cachexia, purulent infection, pneumonia, diarrhea, nephritis, and occasionally through tuberculosis.

Mixed Form. — It must be understood that the two preceding pictures of tubercular leprosy and nerve leprosy are more schematic and didactic than in conformity with the ordinary appearances.

Almost invariably, while one of these groups of symptoms predominates, there occur the most varied associations and combinations. Mixed or complete leprosy is by far the most common, either from the start or more frequently as a result of modifications in the course of evolution; the tubercular form especially tending to become gradually complicated by nervous and trophic disturbances.

Pathological Anatomy.—*Leprous tubercles* or *lepromata* are made up of a sharply limited and very coherent intradermic infiltration, under a normal although stretched epidermis; it is separated from the basal layer by a thin band of normal tissue with a wavy lower border and is prolonged in cuff-form around the vessels, which are affected with endoperivasculitis and around the nerves, the lesions of which are inconstant, as well as around the glands.

The infiltration is made up principally of the "lepra cells" of Virchow, large cells with a clear and vacuolated protoplasm, sometimes polynuclear; furthermore, by connective-tissue cells, a few lymphocytes, mast-cells and occasional plasmocytes; sometimes giant cells are found. Bacilli are more abundant than in any other microbic disease; they are arranged in clusters or bundles, especially

in the lepra- and connective-tissue cells and are sometimes extracellular and agglomerated in a glairy substance (glœa); the name of *globi* is applied to balls or strands composed entirely of bacilli. Caseation is not found in lepromas.

In recent spots or *leprides*, only perivascular cuffs of round cells are found, which in the course of the successive exacerbations become mixed with lepra-cells at first not very large. The progressive increase and confluence of the perivascular infiltrations lead to the transformation of the spots into tubercles. In opposition to prevailing opinions, I showed in 1897 that there are merely differences in degree between these two lesions and that leprides regularly contain, from their onset, a number of bacilli which although not very considerable can nevertheless be demonstrated by a suitable technic. [In 1899, in Unna's clinic, I was able to demonstrate a few bacilli in the apparently normal skin of a leper after a febrile attack.]

The cutaneous manifestations of leprosy result from bacillary embolisms. Several authors have noted a bacillemia at the time of the exacerbations, accompanied by polynucleosis; after the crisis a transitory lymphocytosis follows and then a persistent eosinophilia, which is sometimes very marked (over 30 per cent. according to Gaucher and Renaut).

Leprous neuritis was described by Virchow. When the nerves are involved, they present the changes of parenchymatous and sclerotic neuritis, of peripheral origin and centripetal course, according to Gerlach, Dehio, etc., or with multiple points of attack. Abundant collections of bacilli may be found, especially in the mixed form.

Diagnosis.—The disturbances of the period of invasion are generally not referred to their true cause until after the appearance of more significant manifestations.

The question of a possible leprosy most frequently arises in connection with erythematous or pigmented spots or even of tubercles, which may be solitary or agminated in lupoid or syphiloid patches; in other cases, attention is aroused by nervous disturbances suggestive of polyneuritis, tabes, rheumatism, or especially syringomyelia, progressive muscular atrophy, scleroderma, Raynaud's disease, etc. The features of these disturbances, for instance anesthesia or thermo-analgesia of the spots or tubercles, may sometimes suggest the diagnosis of leprosy from the start. This supposition will be strengthened by knowledge of the patient's nativity or his sojourn in contaminated countries. The important point is to keep leprosy in mind.

The impression received should always be controlled by thorough inquiry and by laboratory tests.

In the first place, the so-called permanent stigmata of leprosy must be looked for; a dusky, cyanotic and pigmented coloration

of the face and the extremities, with softness and dryness of the skin; alopecia of the beard and body, especially alopecia of the external half of the eyebrows; tubercles of the ear-lobule; tumefaction of the nerves, notably the ulnar nerve; atrophy of the muscles of the hand; anesthesias; scars from bullæ on the elbows and knees; leprous orchitis; ocular manifestations such as paresis of the orbicularis, conjunctivitis and episcleritis; rhinitis with hypersecretion and epistaxis, or even perforation of the septum; hoarseness of the voice, etc.

Absolute scientific proof is furnished by the demonstration of the bacilli. These may be found: by biopsy of a leproma or a lepride, in sections or on a smear preparation of a piece of excised tissue; in default of these procedures, in an extirpated gland, with or without the aid of the antiformin method; in the nasal mucus, especially in the tubercular forms after the administration of 4 grams of iodide two days in succession if necessary.

The intradermo-reaction to leproline, which was tried by Mantoux and Pautrier; also, the sero-agglutination proposed by Gaucher and Abrami; as well as the complement-fixation reaction, with a leproma extract as the antigen; all these are undoubtedly valuable methods, but cannot as yet be described as entirely satisfactory. It must be remembered that the blood serum of lepers, even those free from syphilis, usually yields a positive Wassermann reaction and that eosinophilia is common in their blood.

Prognosis and Treatment.—As to the curability of leprosy, it must be remembered that prolonged remissions and arrest of the disease which might be considered as cures, have been observed even in countries where leprosy is endemic.

Moreover, there are incomplete (fruste), benign forms in which the symptoms are reduced to a few spots, circumscribed anesthesias, partial amyotrophies, without any further evolution. A recrudescence, however, is always possible. I have repeatedly observed the redevelopment of a checked leprosy at the time of the patient's return to the land of origin of the disease. In short, the possibility of a cure of leprosy must be conceded, but with reservations.

The *prophylaxis* is more important than therapeutic measures. The rule of compulsory reporting of cases and relative isolation of the patients, although harsh in its application, has yielded such brilliant results in Norway (243 cases in 1902, where there were 2598 in 1856) that it should be enforced in all contaminated countries with modifications to suit conditions. Occupations which involve the risk of the spreading of the contagion must be forbidden to lepers.

Persons who come in contact with lepers must practise measures of scrupulous cleanliness and strict personal hygiene. The lepers must be regularly bathed, cleaned and bandaged. As far as possible,

they must be advised to leave the infected countries and to reside in a healthful temperate climate; under these conditions, lasting improvements and quasi-cures are sometimes observed.

No *medication* possesses an absolutely specific value. Mercury in soluble injections, or in the form of calomel or gray oil, seemed to give good results in my experience and in that of Crocker, Ehlers and Haslund. Ichthyol, salol and salicylates have been recommended. Treatment with arsenobenzol is useless. The classical treatment, the best on the whole, although its value is disputed, is chaulmoogra oil, given in doses of V drops, progressively increasing to CL and CC, in emulsion or better in capsules, for periods of two months, several times during the year. Given by the mouth, it is often not well tolerated; I have always been pleased with intramuscular oily injections according to the formula of Brocq (chaulmoogra oil, 70 cm., eucalyptol 30 cm., in sterilized ampoules of 5 cm.). Its substitutes, gynocardic acid and gurjun balsam, seem to be less active. Opinions differ concerning the value of nastine injections; the name nastine is applied to products derived from cultures of a streptothrix extracted from lepromata by Deycke; these injections have sometimes proved injurious.

[There is no doubt that cases of leprosy have been arrested and possibly cured by chaulmoogra oil. The problem is to introduce the remedy in sufficient dosage. Hollmann and Dean (Honolulu) have recently published some very striking results obtained by subcutaneous injections of ethyl esters of chaulmoogra fatty acids.]

Very hot baths bring great relief and a sense of well-being to lepers, especially in painful cases.

The tubercles break down and disappear on cauterization with the galvanocautery. The ulcers must be kept very clean and covered with moist aseptic dressings; I have obtained good results from daily painting with Mencières fluid [therapeutic notes, section 3]. The ocular lesions are benefited by subconjunctival mercurial injections, atropin and warm local douches.

In a general way, different local measures are required, according to the nature of the lesions and their seat. There is no doubt but that an actively and persistently treated case of leprosy takes an infinitely more favorable course than when the disease is left to itself.

The future undoubtedly belongs to a serum [or chemotherapeutic] treatment which still remains to be discovered.

GLANDERS.

Glanders (French, la morve, German, Rotz) [*Malleus*] is a very grave contagious and inoculable microbic disease which affects soliped animals and is transmissible to man. It takes an acute or a

chronic course. In different cases, the general symptoms, or the visceral lesions or the cutaneous and mucous lesions predominate.

There is no valid reason for maintaining the formerly made distinction between glanders on the one hand, an internal disease affecting the respiratory apparatus and especially its first passages, the nasal fossæ—and *farcy* on the other, an external disease affecting the skin and hypoderm; for their cause is identical.

The frequency of glanders in horses and asses had considerably diminished in France before the war, especially since the discovery of *malleine*, a secretory product of the bacillus, made its early diagnosis possible.

Human glanders, which is rare, is in the vast majority of the cases of direct equine origin, so that those whose occupation puts them in contact with horses are apt to contract it by cutaneous inoculation, without there necessarily existing a demonstrable erosion. It seems probable that infection may also occur through the nasal fossæ.

The bacillus of glanders is slender and shows clear spaces [vacuoles?] it does not take the Gram-stain; it can be stained by Weigert's method or by Nicolle's method with tannin, but easily becomes decolorized. Its cultures on potato assume a tawny, then brownish color and are characteristic. It is very dangerous to handle and can be successfully inoculated into all laboratory animals.

Glanders pus introduced under the guinea-pig's skin gives rise in a few days to a swelling from which culture-material may be extracted; on injection into the peritoneum of a male guinea-pig, it gives rise in three days to a severe orchitis. Straus pointed out the value of the last-mentioned reaction for the diagnosis, but it is not absolutely conclusive. The serum of animals having glanders agglutinates the glanders bacillus at 1 to 500 or 1 to 1000; but agglutinins may also exist in some normal sera. The complement-fixation test is made by means of an extract of bacilli as antigen.

The anatomical lesions of glanders are characteristic only when they are found to consist of glanders granulations; the latter have been compared to tubercles, but are rather minute abscesses, composed principally of polynuclears; they become necrotic and liquefied in their center; a peculiar fragmentation of the nuclei occurs, known as *chromatorhexis*.

The clinical forms are multiple and very dissimilar:

Acute glanders is a blood-infection, a septico-pyemia, which occurs primarily or as the result of cutaneous inoculations (farcy chancre) and sometimes as the termination of the chronic form. It assumes a typhoid or rheumatoid course, or especially that of pyemia, with a remittent, later an irregular fever, great weakness,

arthritis, respiratory symptoms, a grave general condition; it kills in five to twenty days.

Its cutaneous manifestations may consist at the onset in an ascending lymphangitis of the limbs promptly followed by abscesses; or in erysipeloid swelling of the face, of dark red color, with diffuse borders, without elevation, on which blisters or sloughs make their appearance; ultimately, after several days, a disseminated pustular eruption develops, resembling variola but not umbilicated and with a tendency to ulceration and gangrene; sometimes, abscesses appear and as they multiply cover the limbs with so-called "farcy-buds," (acute farcy).

Chronic glanders behaves like a localized infection with a tendency to become generalized in exacerbations. It manifests itself as phagedenic penetrating ulcers and abscesses; these are situated on the face or on the limbs. The clinical picture becomes complicated by general, febrile, articular, testicular, digestive and respiratory disturbances and usually terminates in acute glanders; a cure is rare; as a rule it is merely apparent or temporary. The duration is from eight to fifteen months or sometimes several years.

Mutilating glanders of the face is the most interesting form for the dermatologist. It begins with tuberculo-ulcerative lesions of the nasal fossæ or the buccal mucosa; by extension and by accessory dermic abscesses originating in the vicinity, the ulceration (which has a puckered floor, livid margins and a profuse discharge of yellowish pus) reaches the face, destroys the soft portions of the nose, the cheeks, the lips and sometimes the nasal septum, exposing the bones without attacking them; the glands are usually enlarged. Festooned borders, as if gnawed by the teeth of mice, and pustules or aberrant sloughing ulcers are features which should attract attention. Observations concerning this form of glanders are rare (Hallopeau and Jeanselme, Besnier, E. Hoffmann); probably some cases are not recognized. The differential diagnosis is difficult from tertiary or hereditary ulcerative syphilide; lupus vorax; mycoses; [espundia] and other phagedenic affections. Laboratory tests are required for its confirmation.

Treatment.—There is no specific treatment for glanders at the present time. Treatment with mercury, iodides and arsenobenzol, which have been empirically tried, is without reliable effect. Hence, in cases with a known initial lesion or in chronic ulcers due to glanders, the question of excision arises when this is practicable, or of cauterization with the actual cautery. Radiotherapy might serve to dry out the ulcers and inhibit their progress. The abscesses must be immediately incised, scraped and disinfected. But the prognosis is extremely gloomy.

VERRUGA PERUANA.

Verruga—also named Carrion's disease or Oroya fever—is a severe infectious, frequently fatal, disease, which is endemic in some Peruvian valleys.

It begins with septicemic symptoms, an irregular fever with rheumatoid pains; followed at the end of a few weeks or months by an eruption of numerous scarlet red pruritic miliary elevations, which increase in size and become pedunculated. There are also larger warty or fungoid nodosities which appear in variable number. Whether small or large, the lesions are extremely vascular and bleed readily; they have at first the structure of a granuloma, which becomes areolar through marked dilatation of the blood and lymph vessels. The eruption predominates on the face, the neck and the extensor surface of the limbs; the mucous and serous membranes as well as the viscera may be invaded.

The pathogenic agent reported by Izquierdo (1885) was rediscovered by Letulle and by M. Nicolle; it is an acid-fast bacillus. Others have looked for it in vain and suspect a special paratyphoid bacillus or a protozoön. Many quadrupeds are susceptible to the disease. Infection is supposed to occur through water or through stinging insects. Verruga confers immunity.

No specific treatment for the disease is known.

ANTHRAX—MALIGNANT PUSTULE.

Malignant pustule is the manifestation of a local inoculation with the *bacillus anthracis* discovered by Davaine.

In man, this inoculation almost invariably results from occupational handling of diseased animals, sheep, goats, horses, cattle, etc., or especially of their hides, where the very resistant anthrax spores persist indefinitely. Malignant pustule therefore usually occupies the exposed parts and is observed in shepherds, veterinaries, butchers, tanners, leather-dressers, brush-makers, wool-sorters, workers in horn, etc. [Several cases of infection from shaving-brushes have been reported.] Infection through contaminated flies, often held responsible by the laity, is possible but exceptional.

Comparable at the onset to a flea-bite, on which a vesicle promptly arises, the pustule becomes indurated and presents a brownish or purplish, granular lenticular spot, which spreads and becomes a slough encircled by vesicles. Itching is rather severe. The circumference of the lesion is the seat of a dark red inflammatory edema, sometimes with strands of lymphangitis. The entire region is invaded by a gelatinous infiltration. Finally, the slough is shed, the gangrene increases in depth and in extent, the phenomena of general infection appear; they consist of a high fever, with a weak

irregular pulse, difficult respiration, sweats, hemorrhages and delirium; death occurs in collapse.

The duration of this course varies from twenty-four hours in fulminating cases to twelve or fifteen days. A spontaneous cure is possible, but must not be expected.

The name of *malignant edema* has been employed for a variety in which the central slough is absent; the symptoms consist of a soft swelling with vesicles and of early signs of severe septicemia. Anthrax edema is seen especially on the eyelids or on the lips.

Treatment.—The old treatment, by excision or cauterization with the actual cautery, is at all promising only on a very recent and still doubtful lesion. Repeated injections, around the pustule, of a carbolized or iodine-iodide solution have been recommended; arsenobenzol has been successfully employed. These measures, however, must make way for serotherapy. Anti-anthracic serum, introduced by Marchoux in 1895, since investigated by Sclavo and others, must be employed in subcutaneous injections of about 40 c.c. and repeated several days until the edema diminishes; it will cure malignant pustule provided it is employed at the onset and even sometimes when the blood-culture is already positive.

CUTANEOUS DIPHTHERIA.

Although reported by Chomel and Samuel Bard in the eighteenth century and described by Trousseau, cutaneous diphtheria has become definitely characterized only since the discovery of the Klebs-Loeffler bacillus. It is discussed in numerous publications, of unequal value, the most important being the contributions of Neisser, Schucht, as well as the reviews of Marchalko, Knowles and Frescoln (1914). It must be distinguished from the ordinary eruptions of urticaria, erythema and purpura, which are frequently observed in diphtheritic patients, especially in those who have received serum injections.

Cutaneous diphtheria, due to the growth of the diphtheria bacillus on the skin, is as a rule secondary to involvement of the mucous membranes and the result of auto-inoculation. More interesting, but less common are the cases in which it is primary and exclusively localized on the external integument; these are derived from direct contagion by a diphtheritic patient or a germ-carrier, or from indirect contagion by clothing, bedding, dressings, etc. It must be kept in mind that although diphtheria principally attacks youthful individuals, no period of life escapes the disease.

Symptoms.—Undoubtedly the most common clinical form is that beginning with spots or patches of eczematiform dermatitis, situated at the circumference of the orifices of the mucous cavities in which the diphtheria runs its course, for example under the nostrils,

around the mouth, the eyes or ears, and also around the vulva, the prepuce and the anus, Wounds or even the slightest excoriations, located anywhere, impetigo, herpes, cracks, intertrigo and more particularly surfaces which have been denuded by blisters (the danger of which in diphtheria was emphasized by Trousseau), are also very apt to become the seat of diphtheritic infection.

The infection manifests itself by a serous, turbid or purulent, sometimes fetid exudation with swelling of the affected surface, a purplish reddening and local pain; this surface becomes ulcerated in places; the appearance of a buff-colored false membrane is common, but it may be absent. Extension is rapid at times and sometimes occurs in form of a pseudo-membranous epidermic elevation, with centrifugal extension and polycyclic contours; or it may result from the coalescence of aberrant vesicular lesions, at other times the patch remains stationary. Fever is not constant; glandular enlargement is usually present; lymphangitis in the vicinity or erysipelas are not uncommon.

In the primary cases, the objective features assumed by cutaneous diphtheria are very variable. The most ordinary form is that of impetigo, ecthyma or impetiginous eczema analogous with that described above. In other cases, vesicles have been noted resembling those of varicella or the vesicle of dermatitis herpetiformis (Dawson), or bullæ like those of epidemic pemphigus of the newborn or ulcers with distinctly circinate margins, abscesses and gangrenous patches; exceptionally the lesions may be of different types in the same patient.

In connection with this polymorphism, it is noteworthy that cutaneous diphtheria is often not pure, either because it becomes grafted on a preliminary skin affection which has served as the infection-atrium, or because the diphtheria bacillus becomes associated with other microbes, notably streptococci and staphylococci as is commonly the case. Hence, even since the introduction of serotherapy, cases of cutaneous diphtheria with a fatal outcome are not very rare.

Diagnosis.—Epidemiological factors, or the coexistence in the patient of a lesion of the neighboring mucous membranes, conjunctivitis, otorrhea, rhinitis, erosions of the buccal commissures, angina even if indefinite, vulvitis and balanitis, must arouse attention. The demonstration of a false membrane on a dermic lesion is far from being conclusive. The key to the diagnosis is really furnished by the demonstration of the specific bacillus in smear-preparations, in cultures and by inoculation into guinea-pigs.

The Loeffler bacillus is generally long, straight or slightly curved, in intermingled rods; it is Gram-positive; at its swollen extremities it presents metachromatic polar corpuscles, staining by the Neisser method; cultures on beef serum, at 98°, yield papular colonies of a

grayish white color in eighteen to twenty-four hours; it acidifies glucose culture-media; inoculated into guinea-pigs, it causes death in twenty-four to seventy-two hours.

The *pseudo-diphtheritic bacillus* of Hofmann, from which it must be distinguished, is shorter, ovoid, has no distinct polar bodies, does not acidify glucose media and, most important, does not kill guinea-pigs. The fusiform bacillus of Vincent is Gram-negative.

Treatment.—Even in merely suspicious cases, pending the diagnosis, prophylactic measures for the avoidance of contagion are in order. The injections of antidiphtheritic serum have entirely changed the prognosis of diphtheria; large doses should accordingly be employed. As an accessory measure, the lesions must be carefully cleansed and local applications be made in conformity with the dermatological type and with the supposed microbic associations. The physician will accordingly prescribe for example, in a given case local applications of hydrogen peroxide water, or strong disinfectants; moist antiseptic or rather cytophylactic dressings, or dressings with polyvalent serum, etc.

SOFT CHANCRE.

Soft chancre, *simple chancre*, or *chancroid* [*ulcus molle*] is a specific and contagious ulceration due to inoculation of the bacillus discovered by Ducrey (1899) and investigated by Krefting, Unna, Nicolle, etc. This bacillus is a short rod with rounded extremities, which appears separately, in groups, or frequently in small chains, whence the name of *streptobacillus;* it is stained with carbol blue, dilute Ziehl's solution, etc.; it is Gram-negative; often only its two extremities take the stain. Its culture requires special conditions and was for the first time successfully obtained by Langlet on a medium made of peptonized human skin; Besançon, Griffon and Le Sourd obtained cultures on blood-agar. The streptobacillus can be inoculated into various species of apes. It possesses a striking resemblance to the bacillus of bubonic plague.

Soft chancre almost invariably results from direct venereal contagion, through the deposit of chancroid pus on some traumatic or pathological erosion during sexual intercourse; more rarely it is derived from an indirect contagion.

The frequency of soft chancres in the same country or the same city is subject to very considerable fluctuations. A preceding attack confers no immunity, as illustrated by countless positive inoculations which have been obtained on the same individual. At the time when the single or dual character of chancre virus was still a matter of controversy, erroneous hopes were entertained of a possible vaccination against syphilis by means of this so-called syphilization method.

The bacillus grows at the site of the infection, or inoculation for diagnostic purposes in doubtful cases, the lesions developing without any incubation, they are already characteristic at the end of twenty-four or forty-eight hours or three days at most.

An *incipient chancre* presents the appearance of a vesicopustule with an inflammatory areola; on removal of its covering, a conical ulceration is exposed which penetrates deeply into the cutis. Spontaneous auto-inoculations, notably on the labia majora and in the intergluteal fold often manifest themselves as small perifollicular pustules which have been named *chancroidal folliculitis* or *miliary chancres.*

Papular chancre is a rare variety, but useful to know; it appears as a flattened or slightly acuminate papule with a soft base. Papular chancres are often multiple and may disappear after a short time, or they may become crested with a vesicopustule followed by ulceration. In the folds of the vulva and anus, simple chancres may assume the form of ulcerative and suppurating fissures.

The *mature chancre* (p. 281) rarely attains or exceeds the size of a 5-cent piece. At the end of a period varying from two to six weeks, it loses its virulence, discharges less abundantly, granulates and heals spontaneously. Exceptionally, soft chancres are met with which persist several months without progressing and seem to be of attenuated virulence although still capable of auto-inoculation. Soft chancre always leaves a cicatrix with sinuous outlines, which may be smooth or honeycombed.

No general disturbances from chancroidal infection are known, the disease being purely local. However, various complications may occur. The most common of these is *suppurating chancroidal bubo;* walking, fatigue and absence of local cleanliness are predisposing factors. From the onset, during the course, or even after cicatrization of the chancre, the corresponding gland becomes swollen, the tissues are congested, fluctuation may appear and unless treated, spontaneous rupture takes place through a dark red and thinned skin. The pus of this bubo, contrary to the view of Straus, is virulent from the beginning and contains the bacillus. Spontaneous ulceration of a bubo ordinarily assumes a chancroidal character, that is, the skin becomes detached and sinuous burrows as well as secondary fistulas are formed. This likewise occurs, although less frequently, in case of surgical incision.

Gangrene of the prepuce and a portion of the sheath of the penis is occasionally observed in the course of subpreputial chancres.

At the anus, soft chancre generally gives rise to an elevated condyloma (chancre in the form of pages of a book); it may invade the anal canal (chancroidal anitis of Ravaut and Bord, 1909), causing painful and bloody stools.

Phagedena is a rare but formidable complication (p. 293).

Histology shows soft chancres to consist of a loss of substance of the epidermis and the cutis; it is covered with a layer of pus containing streptobacilli; it presents radiating processes which fissure the floor and the borders. Beyond this layer is a dense infiltration of well developed plasma cells. The bloodvessels show very pronounced changes of endoperivasculitis. The lymphatics are dilated. The ulceration results from a sort of digestion of the tissues under the influence of the parasite.

If the *treatment* of soft chancre is to be efficient, it must not be limited to applying a little iodoform or a caustic agent to its surface. The ulcer must first be cleansed every day and carefully dressed; this is accomplished by means of cotton wipes or rolls dipped in soapy water, alcohol, or benzine and not until then is the alterative topical agent to be applied. The best is iodoform as a powder or iodoform with an addition of camphor (2 to 5 per cent.); it rarely causes dermatitis provided care be taken to deposit it only in the ulceration itself; its persistent and suggestive odor has led many physicians to employ substitutes (iodol, diiodoform, aristol, europhen, airol, etc.), but none is so efficient. The odor of iodoform can be concealed to some extent by an addition of coumarin or even of powdered roasted coffee beans. Almost equally valuable is silver nitrate in aqueous solution (1 to 15), in customary usage; or pure liquid carbolic acid which may be used only with extreme caution; carbolic acid has also been recommended, in alcoholic solution (10 per cent.), as well as ferropotassic tartrate (15 per cent.) and potassium permanganate (from 2 to 4 per cent.). The borohypochlorite powder of Vincent seems to be greatly superior. A small cotton dressing must be applied.

The virulence of the streptobacillus is destroyed by heat, so that very hot local baths should be prescribed, to be repeated at least two or three times a day, or preputial irrigations, or continuous applications of compresses soaked in hot boiled water (42° to 45°). Audry recommends the employment of radiant heat of the thermocautery brought within a few millimeters of the ulceration until its surface is dried out. A superheated air apparatus is still more convenient. This procedure, which should be carried out two or three times, is somewhat painful but very effective.

Suppurating chancroidal bubos are sometimes aborted under the influence of rest in bed, hot compresses and compression bandages. In case rupture appears inevitable, a careful incision may be made or, better, filiform drainage with two threads established; irrigations are applied every day until the discharge becomes merely serous, using a silver nitrate solution (1 per cent.) or a suspension of iodoform. Chancre of the anus is treated by means of wicks covered with a layer of iodoform-vaseline.

CHAPTER XXVIII.

DERMATOMYCOSES.

The name of dermatomycoses must be reserved for diseases developing in the skin or reaching the cutis secondarily and caused by vegetable parasites of a higher order in the scale than the schizomycetes or bacteria. I separate these from the *epidermomycoses* which have already been discussed (Chapter XXV).

After actinomycosis (the first known type in this category) and Madura foot, which is related to it, the investigations of American, French and German observers revealed the blastomycoses, and finally the sporotrichoses, discovered in America, studied more particularly in France in the last ten years and found in nearly all the countries of the earth.

It is an established fact that the place occupied by the mycotic infections in human and animal pathology is decidedly larger than was formerly believed. Their knowledge is not only of scientific interest but also possesses considerable practical and therapeutic importance.

The clinical manifestations of the dermatomycoses are polymorphous and often ambiguous; laboratory investigations, notably cultures, are indispensable for a positive diagnosis and for identification of the pathogenic agent. This explains why our acquisitions in this domain are of relatively recent date and as yet incomplete. Moreover, it has been recognized that almost identical clinical pictures may be produced by different species, while inversely parasites of the same group give rise to dissimilar affections. Accordingly there is no conformity between the pathology and the botanical classification. The latter is moreover still in the tentative stage in regard to the lower fungi and not definitely established.

The following according to E. Pinoy are the botanical groups from which the principal dermatomycoses are derived:

Nocardia (actinomycoses, mycetomas); *Cohnistreptothrix* (actinomycosis); *cryptococcus* (blastomycoses); *oïdium* (blastomycoses); *madurella* (mycetomas); *sporotrichum* (sporotrichoses); *saccharmyces* (blastomycoses); and *aspergillus* (mycetomas).

ACTINOMYCOSIS.

The parasite which causes so-called "lumpy jaw" in cattle, known in France as sarcoma of the bovine maxilla, was named *actinomyces*

by Bollinger. It gives rise in man to suppurating neoplasms and gummous formations. It appears in the pus or in the tissues in the form of yellow granules, from $\frac{1}{10}$ to 1 mm. in diameter, opaque and of oily consistence; they can be seen with the naked eye in pus which has been crushed between two glass slides or diluted with water in a watch-glass. These granules are mulberry-shaped and composed of a fragmented feltwork of mycelium in their center from 1 to 2 μ in width and at the periphery by large club-shaped refractive swellings, resulting from degeneration of the filaments, arranged in contiguous rays (Fig. 176). These clubs are sometimes absent. Unlike the mycelium, they do not stain by the Gram method and are

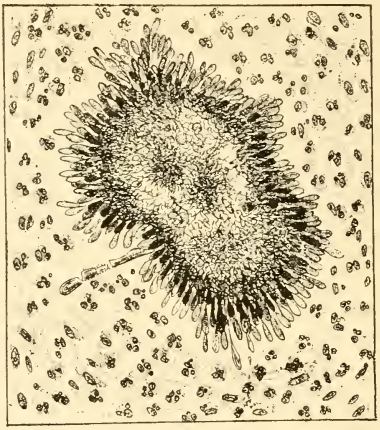


Fig. 176.—Actinomyces granule. After Pinoy, Bull. de l'Inst. Pasteur, November 15 and 30, 1913, xi.

not present in the cultures. The latter show that two distinct species may be involved, differing in their cultures; either Nocardia bovis, growing readily, aërobic, not inoculable into animals; or Cohnistreptothrix israëli, anaërobic, growing with difficulty and inoculable into the peritoneum of guinea-pigs and rabbits.

Actinomyces live quite abundantly as saprophytes outside of the animal tissues. Man is rarely attacked through contagion from herbivorous animals; as a rule he becomes infected in the same way as cattle, notably through grains of cereals which have wounded the skin or the mucous membranes or been carelessly swallowed. The habit of chewing herbs or bits of straw when strolling through the fields must be regarded as dangerous from this point of view.

Actinomycosis exists in all countries. In France, its relative frequency in the vicinity of Lyons was pointed out by Poncet, in the region of Bordeaux by Petzes. It is more common in Germany [and rather rare in America].

Clinical Forms.—*Cutaneous* actinomycosis, which alone is here considered, may be primary or secondary; that is, the skin affords the parasite an avenue of entrance which is rare, or of exit, which is frequent. The lesions are cervico-facial in at least two-thirds of the cases, or they may be thoracic or abdominal, or located at the anus or on the extremities.

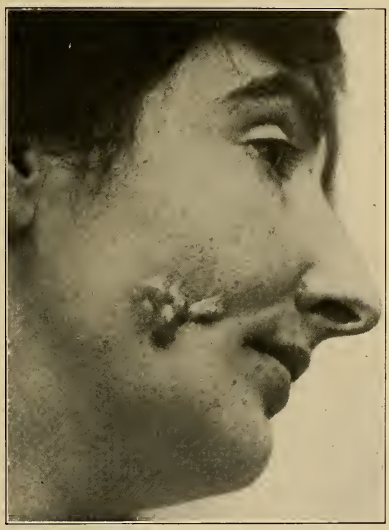


Fig. 177.—Actinomycosis of the cheek.

At the onset there is a hypodermic nodosity, with a rose-colored, hardly painful deeply adherent surface; the center of the mass promptly softens and becomes fluctuating; the purplish skin gives way and permits the escape of a small amount of purulent or sanious fluid, containing yellow granules. At the same time, other nodules have formed nearby which collect into patches and follow the same course; the ulcerations remain fistulous and granulating. (Fig. 177).

There is presumptive evidence of actinomycosis when lesions presenting the following clinical features are encountered: nodosity

followed by a conglomerate tumor of woody hardness, often deeply adherent, with a purplish surface, containing foci of a slowly gathering gummous pus; absence of corresponding glandular enlargement; tendency of the newformation to invade all tissues indiscriminately, the muscles, the vessels and even the bones.

These characteristic features usually suffice for the differential diagnosis from dental abscesses, chancroids, lupus and tuberculosis verrucosa, tuberculo-gummous syphilides, epithelioma, sporotrichosis, etc. The demonstration of the *yellow granules* will supply the necessary confirmation. Sero-agglutination and the fixation test have yielded results, but these are not reliable.

Pathological Anatomy.—The parasite stimulates leukocytosis and proliferation of the fixed constituents in the form of nodules. The latter are accordingly formed in their center of an actinomyces granule in an amorphous necrotic zone, often surrounded by a wreath of giant cells; next by a zone of plasma or epithelioid cells; at the circumference there is a more or less considerable infiltration of leukocytes and swollen connective-tissue cells which insinuates itself between the connective-tissue bundles. The last-mentioned zone is of fibro-sarcomatous appearance and of a hard or lardaceous consistence. The vessels are often intact.

Treatment.—Medication with potassium iodide, introduced by Thomassen, has markedly improved the otherwise very gloomy prognosis of actinomycosis. Failures do occur, however. The customary dose is at least 6 grams daily; Pinoy recommends in addition a salt-free diet and multiple deep cauterization of the lesions. In recent and not very extensive cases, a cure may be obtained in a few weeks. Local iodide injections are also administered. Radiotherapy seemed to me to exert an evidently favorable effect.

Long-standing, deep and complicated cases require daily doses of 6 to 12 grams of iodide and moreover the surgical removal or curetting of the foci.

MYCETOMA OR MADURA FOOT.

This disease, which is endemic in India, Western Africa, Morocco, etc., is due to several species of mucedineæ, which penetrate into the organism by means of a foreign body such as thorns or splinters of wood, especially in bare-foot natives. The forms of *nocardia, madurella mycetomi* and *Tozeuri* have been recognized; Ch. Nicolle grew cultures of an *aspergillus* inoculable into pigeons (Pinoy). An analogous affection has been observed in America.

Beginning on the sole of the foot, nodosities make their appearance which soften and discharge a sanious fluid containing granules of parasites; these granules are white, red or black, which has led to

three varieties of mycetoma being described. They consist of a mycelial feltwork without clubs. Through the growth and multiplication of the nodes, the foot becomes deformed, globular and assumes an elephantiastic appearance while covered at the same time with bullæ or tubercles and hollowed by fistulous tracts; the leg on the other hand becomes wasted and atrophic. The glands are rarely enlarged.

Iodide is not particularly successful and must be reinforced by a salt-free diet and cauterizations. The condition is relieved by the hyperthermic baths advocated by Legrain. Curative treatment must be surgical.

BLASTOMYCOSES.

Under this heading diseases are classified which are due either to yeasts or *saccharomycetes* or to parasites belonging to other botanical groups but which under certain conditions present themselves in the form of proliferating buds like the yeasts; the exact determination of the latter has not always been possible.

The clinical appearance of the blastomycoses is very polymorphous. To illustrate these cases, I shall limit myself to outlining the symptomatology of two of the best known types of the disease:

1. *Busse-Buschke Type.*—This extremely rare type was very thoroughly investigated and described by the authors whose names it bears; the cases of Ormsby-Miller, Curtis and Hudelo are apparently closely related to it. The pathogenic agent is probably a *saccharomyces.*

The condition at the onset is marked by osseous and articular lesions; later on, or sometimes from the start, follicular inflammations develop or more often disseminated gummous nodules which become transformed into ulcers; their purulent contents and fungosities contain yeasts capable of cultivation and inoculation. The disease is febrile, taking a rather rapid course; the general health is gravely impaired. Death results from glandular and visceral lesions.

2. *Gilchrist Type.*—This form is said to be less uncommon; it was pointed out by Wernicke (1892), then by Gilchrist and Ricketts (1896) and was at first attributed to protozoa. About forty observations have been published, the majority in America (Hyde, Montgomery, Stelwagon, etc.), and only five or six in Europe. The name of blastomycetic dermatitis is often applied to it. These dermatitides are due to two parasites which present a different appearance in the sections: an *oïdium Gilchristi* in form of a yeast and a parasite with multiple external buds, *coccidioides immitis.*

The lesions are dermic and begin on one of the extremities or on the face. A hard, reddish, superficial nodule becomes crusted with a

yellow point and is transformed into a pustule discharging a thick viscid pus. The miliary abscesses increase in number, extend and give rise to an irregular, circumscribed papillomatous, erosive patch, infiltrated but movable on the deeper layers. The vegetating surface is interspersed with miliary abscesses, sometimes covered with crusts and may become partially cicatrized. The lesion extends superficially and the foci often multiply. The duration has frequently been from two to three years or longer. Spontaneous cure may occur (Figs. 178 and 179).

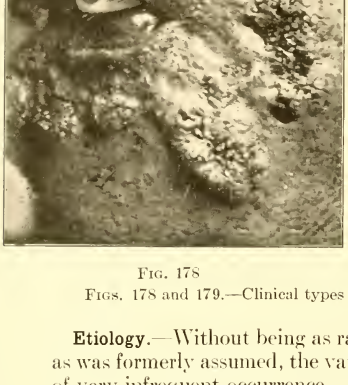


Fig. 178

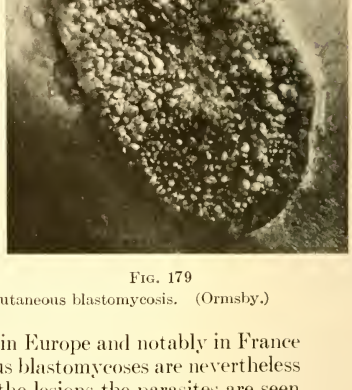


Fig. 179

Figs. 178 and 179.—Clinical types of cutaneous blastomycosis. (Ormsby.)

Etiology.—Without being as rare in Europe and notably in France as was formerly assumed, the various blastomycoses are nevertheless of very infrequent occurrence. In the lesions the parasites are seen as rounded bodies, measuring about 10 μ, composed of a membrane with double contours and granular contents. They are more or less abundant in the sections and may be stained by the Gram-Weigert or other methods. Pus-smears should be washed in ether and examined in a solution of potash.

The cultures in some cases grow readily on various media, notably on peptone-agar with glucose or maltose, preferably in the incubator; in other cases, they are only obtained with difficulty (Gilchrist type). The appearance of the colonies is variable and not very characteristic. The saccharomyces remain in the state of spherical or budding bodies of 2 μ to 20 μ diameter; the oïdia form mycelial-filaments in their cultures.

Inoculation of some yeasts is successful in mice, young guinea-pigs and sometimes in dogs, yielding a sort of pseudotuberculosis.

Man can certainly be infected through the skin and perhaps also in other ways. It must not be overlooked that various species of mucedineæ may be found accidentally on the skin or may live there as saprophytes; they may secondarily infect pathological lesions of various kinds. Great caution is therefore necessary in the interpretation of their pathogenic value.

The several serodiagnostic tests which have been tried do not yield constant or altogether reliable results.

Pathological Anatomy.—In the first-named clinical type, there exists a granulation-tissue rich in vacuolated giant cells packed with parasites.

In the pustulo-vegetative type, intra-epidermic abscesses are demonstrable and in the cutis a superficial infiltration is seen formed by various cells, notably plasmocytes with rare giant cells. The proliferating epidermis sends branching proliferations hollowed by miliary abscesses into the depth of the tissue. In a general way, the appearance resembles both tuberculosis and epithelioma. The parasites are scattered and are found especially in the abscesses.

Diagnosis.—Vegetative syphilides are less irregular in form and have fewer miliary abscesses. Papillary epithelioma has a hard consistence, friable proliferations and whitish masses formed of epithelial cells. The resemblance to tuberculosis verrucosa may be perfect; Gilchrist described his case as pseudolupus verrucosus. The blastomycoses are perhaps less purplish and less painful, more extensive and with more numerous foci.

In suspected cases, aside from examinations of the pus, biopsy and cultures, it is advisable to inoculate guinea-pigs for the discovery of tuberculosis and to perform the Wassermann test for the elimination of syphilis.

Treatment.—The blastomycoses are grave affections requiring energetic treatment. Iodides have proved remarkably successful but must be given in large doses, from 6 to 8 grams daily and long continued. Local surgical treatment is sometimes imperative. Dressings with iodine solutions should be applied.

SPOROTRICHOSES.

The sporotrichoses are by far the most frequent dermatomycoses.

For the first reported cases credit is due to Schenk (1899) and Hektoen and Perkins (1900); but for our knowledge of the mycoses in general and sporotrichosis in particular in all its forms we are indebted mainly to French contributions, particularly those of de Beurmann with Ramond (1903) and especially with Gougerot (1906–1912) whose work drew attention to this subject.

The *sporotricha* are mucedineæ, namely lower filamentous fungi.

with a creeping, regular, branching, partitioned or continuous mycelium bearing numerous short sporulated branches. The spores, measuring from 3 to 6 μ originate separately or in groups of two, either on the filaments or more abundantly on the conidiophorous branches. It is not known if these parasites do not possess higher fructification-forms.

Among the pathogenic species, the best known and the most frequent is the *sporotrichum Beurmanni*, which I shall utilize as the type, the sporotrichum Schenckii differing from it in its faintly colored or white cultures and various other features; the sporotrichum Dori is readily distinguished from it.

The *culture* method is the procedure of choice for the demonstration of these organisms; the technic of de Beurmann and Gougerot is simple and easy. Streaks of the pus on unsealed peptone-glucose agar tubes at room temperature yield at the end of six or eight days visible cultures which by the twelfth day have become exuberant and are often pure from the start. The method of Gougerot, which consists in dropping some of the pus on the glass of the tube or on the border of the agar, often permits a rapid diagnosis, in two or three days, by the demonstration of small gray filamentous stars, visible to the naked eye, or with the microscope through the culture tube; incubated at 98° the growth is less abundant. Other culture-media, notably carrots with glycerine, may be utilized. The whitish and acuminate colonies gradually turn brown, spread out and convolute on their margins, which are surrounded by a flat, finely radiating areola. These chocolate-brown cultures are characteristic.

Inoculations into animals have yielded inconstant results; the virulence is slight. De Beurmann and Gougerot have obtained systemic infections; mice are the most suitable animals.

The portals of entry in man escape observation; sometimes a previous traumatism has been noted at the affected point; infection seems to occur especially by way of the mouth. [A laboratory worker in Chicago was accidently infected on the conjunctiva.]

Symptoms.—The sporotrichoses are in the highest degree syphiloid or tuberculoid. However, their tissue and their pus are not inoculable into adult guinea-pigs, which are susceptible to tuberculosis; they are not influenced by mercurial treatment; left untreated, they persist and multiply, whereas they are often readily curable by iodide treatment; finally, they contain a special parasite. This combination of features suffices for their recognition as a distinct entity.

The disease is extremely polymorphous in its manifestations; the following are the principal clinical types:

1. The *disseminated gummous form;* the gummas (p. 270) may be softened but not ulcerated; or ulcerative, ulcero-vegetative, ecthymatiform; they may assume to the highest degree a syphiloid or

tuberculoid appearance; or in other cases that of large more or less multiple abscesses.

2. *Lymphangitic form* (Fig. 180), consisting of lesions of the preceding type in chain-like arrangement above an initial "sporotrichotic chancre;" this form is encountered especially on the limbs and sometimes on the head.

3. *Extracutaneous forms*, affecting the bones, the synovial membranes, the testicles and the viscera; it is especially useful to be familiar with primary sporotrichotic osteitis which has been not infrequently observed, for example on the calcaneum; it gives rise to bone-liquefying abscesses and then to fistulas with secondary cutaneous lesions.

FIG. 180.—Sporotrichosis, lymphangitic form; of two and a half years' standing; positive culture; cure in three weeks by potassium iodide.

4. *Ulcerative forms of the mucous membranes*, localized in the mouth or pharynx, may extend to the base of the tongue, the larynx and the trachea; these cases are rare but extremely grave. Sporotrichotic ulcers of the mucous membranes are distinguished by their prominence, their dirty yellowish-gray color, the absence of false membranes on their surface, their tendency toward diffusion rather than deep destruction and mutilation.

Sporotrichotic cicatrices resemble those of syphilitic or tuberculous ulcers; their borders are often irregular and ragged, sometimes showing loosely joined tongue-like processes.

Diagnosis.—The clinical picture may furnish highly suggestive evidence; in the St. Louis Hospital, sporotrichosis is usually recognized at first sight. The diagnosis is based on: the multiplicity and polymorphism of the lesions, which taken as a whole justify a diagnosis neither of tuberculosis nor of syphilis; their more acute course than that of the cutaneous tuberculoses; the viscid whitish pus which escapes from the softened nodules; the inconstancy of

glandular enlargements; and the preservation of good general health.

Two scientific diagnostic procedures are available, however, which are indispensable in doubtful cases, which are very common: (1) Culture, which requires from eight to twelve days; (2) sporo-agglutination, discovered by Widal and Abrami, which furnishes immediate information; it is of value only when positive and at a very high ratio, at least 1 to 200. The serum of patients suffering from actinomycosis or thrush, etc., may also agglutinate a sporotrichum culture, but at a greatly lower ratio.

The reactions after cutaneous or subcutaneous injection of sporotrichum emulsion may confirm the diagnosis, but do not establish it by themselves alone, on account of the possibility of associated reactions due to other mycoses or even to the presence of simple saprophytic yeasts in the patient's throat. It goes without saying that a negative Wassermann reaction, proving the probable absence of syphilis and the negative outcome of inoculation of diseased tissue into guinea-pigs, showing the absence of the Koch bacillus, are signs indirectly capable of corroborating a suspicion of mycosis, but insufficient for the determination of the special agent.

Pathological Anatomy.—The histological lesions of the sporotrichoses are not more characteristic than their clinical appearance. They consist of a nodular inflammation with a suppurative center.

According to the general formula of Gougerot, the sporotrichotic nodule is formed by three concentric zones: at the periphery it is syphiloid, due to its subacute perivascular inflammatory character, with inconstant connective-tissue reaction and mononucleosis with plasmocytes; the middle zone is tuberculoid, through its epithelioid tubercles arranged around giant cells; the center is suppurative, with polynuclear and macrophagic cells. The majority of the tubercles originate from a proliferation of the vascular walls.

One must not expect to find branching and spore-bearing mycelium in the sections or in the pus; the only findings, which are moreover not constant, are short filaments or rather "navicular" bodies, free or contained in phagocytes.

Prognosis and Treatment.—Left untreated, the sporotrichoses persist and their foci increase in number. Under the influence of iodides they are curable in the vast majority of the cases in the space of a fortnight to two months. The non-ulcerated forms subside more rapidly than the others. The existence of a tuberculous or other cachexia, to which the sporotrichosis may be secondary, of course aggravates the prognosis. The case observed by Letulle-Debré [pharynx, etc.] terminated in death.

Potassium iodide should be administered in daily doses of 5 or 6 grams, or more if tolerated by the patient; it is advantageous to

combine with it a salt-free diet; other iodides or injections of iodized oil may be utilized as substitutes in case of intolerance. Locally, it is necessary to puncture the purulent collections and to inject them with a 1 per cent. iodo-iodide solution; the ulcerations should be dressed with the same solution. The treatment should be continued even after an apparent cure has been obtained and be renewed on the slightest threat of a relapse.

CHAPTER XXIX.

INFECTIOUS DERMATOSES DUE TO PROTOZOA.

Up to a few years ago it was assumed that all infectious diseases were caused by bacteria or by lower fungi closely related to the Schizomycetes; those in which the organism had not been found were attributed to unknown bacteria.

The discovery of the trypanosomiases, then of the parasite of syphilis, the other spirochetoses, leishmanioses, etc., has opened a new chapter, to which in all probability additions will be made in the near future.

SYPHILIS.

Syphilis—*lues venerea, pox*—is a systemic infectious disease, transmissible by contact and congenital, due to the *Spirocheta pallida.*

It is extremely contagious and consequently very widely distributed; it may affect all organs and all tissues without exception; give rise to symptoms of the greatest gravity such as cerebral syphilis, tabes, general paralysis, etc.; and be followed by cancer of the mouth, abortion and a high infantile mortality. Thus it is only too true that syphilis is one of the greatest scourges of the human race.

Infection usually takes place through the skin or through the mucous membranes and its most characteristic manifestations are exhibited on the external integument. Their description must therefore necessarily figure in a text-book of dermatology. The general picture of the disease will be given only in brief outline.

Etiology.—In May of 1905, T. Schaudinn and E. Hoffmann reported the discovery in the contagious lesions of syphilis and in the lymph glands, of a parasite which seemed to be the long-sought causative agent of this disease. This has been definitely established by their later studies and by innumerable control investigations. The responsible organism had been seen a few years previously in a chancre, by Bordet and Gengou, who were not able, however, to follow it in the various syphilitic lesions.

The parasite of syphilis, now classified under the name of *Spirocheta pallida* or *Treponema pallidum* is a protozoön of spirillary form, whose cylindrical body, of an average length of 6 to 14 μ and a width of at most 0.3μ, describes close-set narrow spirals, from six to twenty in number; it terminates at both ends in an extremely slender flagellum. An undulating membrane has so far not been

demonstrated. Living specimens on ultramicroscopical examination are seen to move actively for several hours. Some deviations from this typical appearance have been observed, possibly corresponding to developmental stages in a still unknown evolution. The treponema for a long time was refractory to culture in any artificial culture-medium; Schereschewsky, W. H. Hoffmann, Sowade, Noguchi and others succeeded in growing it in pure culture under special conditions.

The agent of syphilis can be stained with less difficulty than was originally believed. It is readily obtained in very thin smears of serous exudate or tissue-juice, fixed by heat, washed repeatedly for three or four minutes with a boiling mixture of the following composition: To 9 c.c. of distilled water add 2 drops of a 1 per cent. solution of potassium carbonate, and 2 drops of neutral glycerine; heat to the boiling-point and add 10 drops of Giemsa's eosine-azure; the specimen must be very carefully rinsed in running water before it is dried and examined. Burri's method is also very convenient: the exudate is mixed on the slide with a small drop of India ink, spread out in a very thin layer, allowed to dry in the air and then directly examined in immersion oil; the parasites stand out white on the dark background. In the tissues the parasite is demonstrated by means of the silver reduction methods devised by Levaditi.

The spirochetes are constantly present in very large numbers in the chancre. They are also found in the glands and in great abundance in mucous patches and recent papules of all kinds; they are less frequent in the roseolar spots, a blister produced by the application of a fly-plaster to a papule often contains many spirochetes. Spirochetes have been found in the spleen (Schaudinn), in the suprarenal capsules (Jacquet and Sézary), and in the meninges. Their usual presence in the blood during the active stages of the disease has often been demonstrated, especially indirectly; in the cerebrospinal fluid, the seminal fluid, the milk and the urine, the parasite of syphilis is found only under special and exceptional conditions. They are very rare in tertiary lesions, but Noguchi and several investigators after him found spirochetes in the brains of patients who had suffered from general paralysis. They are very abundant in children and fetuses with congenital syphilis, notably in the liver, the spleen, the suprarenals, the lungs, the blood, as well as in the cutaneous lesions. The parasite has been successfully followed in serial inoculation into monkeys and more recently also other animals.

Syphilis was for a long time considered as absolutely peculiar to man; it was taught that neither race nor age nor sex confer immunity against it, but that a first attack renders the patient permanently immune. More recently it has been recognized, however, that the immunity acquired by a first infection may not always be absolute

and permanent; the possibility of reinfection has been demonstrated in some cases, not numerous but convincing.

On the other hand, two years before the discovery of the spirochete (July 28, 1903) Roux and Metchnikoff showed that syphilis is inoculable into anthropoid apes; Lesser, Neisser and many others confirmed these observations. Since that time it has been recognized that inoculation is also successful in the lower monkeys; an attempt has been made to utilize this fact for diagnostic purposes, a procedure which actually possesses but small practical value. More recently syphilis has been successfully inoculated into rabbits, dogs, guinea-pigs and sheep. Inoculation into the rabbit's cornea (Bertarelli) or on the scrotum of this animal (Parodi) has so far yielded most positive results; their proportion is increased in serial reinoculations and sometimes symptoms of generalization have been noted. *Experimental syphilis* has already led to valuable scientific findings and is sure to furnish a further abundant harvest.

According to the classical teachings, there are two modes of contracting syphilis: by contagion or by heredity.

Acquired syphilis results from a venereal or accidental contact, which may be direct or indirect; *congenital syphilis* is present at the birth of the child and is derived from its parents.

Acquired Syphilis.—The course of acquired syphilis is subject to certain laws. The infecting contact is followed by a latent period known as the first incubation, usually lasting twenty-five days, sometimes shortened to ten or fifteen days or very exceptionally prolonged to sixty and even to ninety days. Then the primary manifestation makes its appearance at the point of the inoculation itself, as the *syphilitic chancre* with which at the end of about a week a satellite *bubo* becomes associated, constituting the primary stage.

Next follows another latent period, the second incubation, of an average duration of forty-five days, at the end of which the *secondary symptoms* appear; these are of various forms, scattered and profuse, usually showing a benign behavior and located especially on the skin and the mucous membranes; as a rule they recur during several months, or sometimes during two or three years or longer when the disease is left to itself or is insufficiently treated.

Later on, especially after the fourth year, sometimes earlier, and often at remote dates of ten, twenty or thirty years or still later, the *tertiary symptoms* may appear. While they have less tendency to diffusion than the secondary symptoms, they cause much deeper damage to the tissues in which they are situated. They may affect any organ, any apparatus, including the skin and the mucous membranes and notably the nervous system, the last-named localization being especially formidable.

The primary lesion, the chancre, is practically never absent, but it may remain undetected, this being actually frequent in women. The few known cases of "immediate syphilis" (d'emblée) or "decapitated syphilis" in men are explained by very unusual conditions. In the absence of early and energetic treatment, the secondary manifestations are only very rarely absent. This remark does not apply to the tertiary symptoms, which develop preferably in tainted, intoxicated or exhausted individuals under bad hygienic conditions and especially in incorrectly or insufficiently treated patients.

It is noteworthy that the secondary and tertiary periods are not always distinctly separated, neither in time nor by the character of the associated manifestations, but may widely encroach upon one another [or overlap]. This subdivision is nevertheless justified by the course of the majority of the cases and, moreover, is convenient from the didactic point of view.

Syphilis may finally eventuate in remote sequelæ which are not influenced by antisyphilitic treatment. A. Fournier designated these as *parasyphilitic symptoms* and interpreted them as indirect consequences of syphilis. This group (general paralysis, tabes, leukoplakia, aneurysms, etc.) will undoubtedly have to be rearranged, for the presence of spirochetes in the brain of paretics shows that in these cases at least, genuine syphilis is responsible [and this is certainly true also for tabes and aneurysm].

Among all the symptoms of syphilis, only the primary chancre and the secondary or tertiary lesions involving the skin and the mucous membranes enter into the scope of this book. These secondary and tertiary cutaneous or mucous manifestations are generally designated under the name of *syphilides*.

Syphilitic Chancre.—Also known as *hard chancre* [ulcus durum], *primary lesion, initial sclerosis*. The syphilitic chancre develops at the point which has served as the infection-atrium of the virus. It suffices that living treponemata derived from a contagious syphilitic lesion be deposited upon a traumatic or pathological lesion, ulceration, herpetic erosion, fissure, or even a trifling excoriation, to bring about the infection; it is not probable that they can pass through the intact epidermis, but they may possibly pass through the intact epithelium of mucous membranes. Transmission usually takes place directly, through sexual intercourse, kissing, accidental or occupational contact; much less commonly through the intermediation of some contaminated object.

During the *first incubation*, the lesion which has served as the infection-atrium has usually had ample time to disappear. The parasite multiplies locally at first without producing a demonstrable reaction and already begins to spread in the organism through the lymphatics and the veins; indeed, an early excision

of the incipient chancre, for the purpose of aborting the syphilis is almost invariably unsuccessful. The production of immunity is not immediate, however; the chancre is often auto-inoculable during the first eleven days following its appearance, according to Queyrat; this would serve to explain the cases of successive multiple chancres.

An incipient chancre, better known through experimentation on monkeys, presents the appearance of a very small slightly papular red spot, or in other cases a scaly crust covering a superficial erosion. The erosion and induration keep on progressing until in a few days the primary sore (Fig. 181) has assumed the six following characteristics, as clearly pointed out by A. Fournier:

FIG. 181.—Syphilitic chancre of the sheath of the penis, of five weeks' standing; untreated.

(1) It is a slight erosion, generally of the size of a dime [about 1 cm.], not an ulceration; (2) it has a round, regular, orbicular form; (3) without marked borders, that is, without prominence, perpendicular depression or detachment, its surface being on the same level as the surrounding tissues or sometimes slightly convex or depressed; (4) of a color varying from red, flesh-color with smooth, moist and glazed or finely granular surface, to a grayish color with diphtheroid surface scattered with ecchymotic points and sometimes covered with a thin brownish crust; (5) an indurated base, which is recognized by grasping the chancre between the thumb and index finger, across its diameter and slightly raising it; thereby demonstrating a characteristic, circumscribed and dry hardness, of very variable thickness, sometimes superficial, like parchment or paper, in other cases deep, resembling pasteboard, or nodular; (6) the chancre is accompanied by a satellite bubo.

This bubo, which according to Ricord's expression follows the

chancre as the shadow follows the body, has its seat in the glands corresponding to the lymphatic territory of the chancre. It consists of a glandular constellation, namely a group of hard, ovoid, movable, painless and non-inflammatory glands; one or two of these glands are apt to be larger than the rest and may present a very evident protuberance. The bubo appears from six to ten days after the chancre and survives it as a posthumous witness for a number of months.

The chancre itself heals in a fortnight to six weeks; the induration as a rule persists for several months, sometimes actually increasing; a cicatrix is seen in only about one-half of the cases.

Exceptionally, an erosion known as *chancre redux* may reappear at the same point, after a very variable period.

The *varieties* of syphilitic chancre are innumerable; but the typical form is by far the most common.

There occur *dwarf* lenticular chancres, or *giant* chancres; *papular, hypertrophic*, or markedly *ulcerative* chancres, or *echthymatous* chancres covered with a fairly thick crust. Although hard chancre is, as a rule, *solitary, multiple* chancres to the number of two or three, or more, are encountered nearly as frequently and up to fourteen have been counted on the same patient; they are simultaneous or *successive*, which may be due to a variable incubation, successive contaminations, early auto-inoculations, or to a chancrous lymphangitis which has given rise to local erosions.

Complications.—The onset of the chancre may be directly preceded by a herpetic eruption, a common and serious source of error; this *herpes* runs the ordinary course, but one or several of the erosions become indurated, thereby revealing their chancrous character. The chancre is not uncommonly accompanied by a local and voluminous first soft then hard *sclerotic edema*, notably of the vulva or the prepuce (phimosis), which suggests its presence, although it helps to conceal it; the chancrous induration can be felt with the finger and confirms the diagnosis. The indurated chancre becomes *inflamed* after traumatism, improper treatment or secondary infection; it becomes painful, bleeds and suppurates; sometimes, the bubo itself undergoes a usually sluggish purulent disintegration. Superficial or penetrating *gangrene* and phagedena are rare complications. *Mixed chancre*, propounded by Rollet (Lyon) is more or less rare according to the environment and results from inoculation at the same point with the Ducrey bacillus and the treponema, their incubation being of very unequal length. When the infections are simultaneous, a soft chancre develops, then becomes indurated and opens again in case it was cicatrized; when the infection is successive, a soft chancre becoming grafted on a syphilitic chancre, it forms an ulcer in the preëxisting induration.

Localization.— *Genital chancre* in men is more frequently located in the balanopreputial groove and the sides of the frenum, but may occur on any other point of the penis, the scrotum or the pubis. Chancre of the meatus and intra-urethral chancre, which are rare, manifest themselves by a serous oozing and a circumscribed induration. In women, the labia majora and minora, the fourchette, the clitoris, and more rarely the meatus, are the seat of the chancre; hardly ever the vagina. Chancre of the uterine cervix would appear less rare if it were more frequently looked for; it presents itself in the form of a distinctly limited red or grayish erosion with a narrow red margin and an induration perceptible to the touch. [In rare cases it may become the site of cauliflower excrescences on the cervix.]

Extragenital chancre is localized on the head in two-thirds of the cases, preferably at the mouth and especially on the lower lip; when it straddles the free borders of the lip, it is crusted in its cutaneous portion, erosive in its mucous portion. After chancre of the lips come in order of frequency, chancre of the tongue and chancre of the tonsils. The latter, which must not be confused with angina, nor especially with the fusospirillary ulceration of Vincent, is unilateral, erosive or ulcerative, nearly always diphtheroid, of woody hardness to the touch, gives rise to only slight pain, persists during four or five weeks and is associated with a usually enormous enlargement of the retromaxillary glands. Chancres of the chin, the eyelids, the conjunctiva, the nostrils, the gums, the scalp, etc., present no very specific features; [the associated adenopathy usually creates a suspicion of their nature].

On the upper limbs, chancre affects chiefly the fingers, the circumference of the nails or the joints; it resembles a badly healing panaritium or a proliferating wound. This localization is not uncommon in physicians and midwives and the same is true for chancres of the eye. Vaccination chancre has become extremely rare since vaccination from arm to arm has been generally abandoned.

Chancre is also frequently situated on the breast, notably in wet nurses.

It is fairly common at the anus in both sexes and is here apt to assume the so-called book-leaf form. There is no region of the body in which its occurrence has not been noted.

Extragenital chancres are usually the beginning of innocently acquired infections, *syphilis insontium*, comprising occupational syphilis, workshop epidemics (glass-blowers, for example), family epidemics and so forth.

The frequency of extragenital chancres as compared to that of genital chancres is in the proportion of 1 in 8 or 9. The possibility of this occurrence must always be kept in mind so as to guard against error, which may have very serious sequelæ.

Diagnosis.—In some cases the diagnosis of syphilitic chancre is evident from the start; in others, the objective features of the lesion merely justify a suspicion which requires to be confirmed; it is well to remember the possibility of chancre no matter what may be the patient's social standing or age or the seat of the suspicious lesion (extragenital chancres).

Even when the diagnosis is certain, it is advisable for reasons readily understood, to state the truth only guardedly and with the greatest circumspection to the patient. Where there is the least doubt, and in fact *in all cases*, it is important to control the objective diagnosis by what may be called the elements of the general diagnosis of syphilis and especially by laboratory investigations. It would be a serious error to begin specific treatment before having absolutely proved of the existence of syphilis.

The history, statements of a suspicious contact three or four weeks before the appearance of the chancre and confrontation in certain cases, possess only the value of highly probable arguments.

The best and most practical procedure for scientific demonstration consists in the ultra-microscopical examination of the serous fluid which has been squeezed out of the chancre after superficial scraping.

For this examination to be valid, the patient must not yet have received specific treatment and no antiseptic or caustic application of any kind must have been locally employed in the last five or six days. The discovery of the characteristic spirochetes is conclusive; in case of failure, the examination should be repeated after a few days' interval [or should be repeated daily if necessary, the patient meanwhile covering the lesion with a moist dressing of normal saline solution].

The Wassermann reaction does not become positive until about the eighteenth day of the chancre.

In Chapter XV (*passim*) may be found the essentials of the differential diagnosis of hard chancre and other analogous ulcerations. Briefly stated, the lesions of the genital region which most frequently lead to confusion with a primary sore are the following:

Traumatic ulcerations are of more irregular form, non-indurated, without bubo.

Herpes may mask an incipient chancre and must be watched; when it has been cauterized, treated with an irritative lotion, for example with perchloride of mercury, or with certain powders such as aristol, the herpetic erosion hardens, the glands become swollen and the aspect may be to the highest degree suggestive of chancre. After several days when the inflammation has subsided under the influence of rest, baths and moist dressings, the artificially induced difficulty will generally have disappeared; if necessary, an ultramicroscopic examination can be carried out.

Soft chancre, with its perpendicular borders and its irregular and suppurating floor, rarely causes difficulties; all doubts can be settled in forty-eight hours at most, by the discovery of the Ducrey bacillus, or better by auto-inoculation in the deltoid region. In case of mixed chancre, this auto-inoculation would be positive and the discovery of the spirochete practically impossible; under these difficult conditions, the appearance of a positive serum reaction often becomes the only practical way of settling the diagnosis.

The name of *chancriform syphiloma* is applied to a lesion which may appear between the second and the fifth year, preferably at the point where the original chancre was located, the features of which it may closely reproduce; or it may be more irregular in contour and deeper. In all cases, there is no bubo, the Wassermann reaction is positive from the start and the antecedents are sufficient to post the physician. The interest of this manifestation lies in its frequently having given rise to the suspicion of a reinfection; it has been thought that it might sometimes be the result of a specific reinoculation on an imperfectly immunized territory.

The *treatment* of syphilitic chancre must be very simple; it is essential to avoid all irritation.

Early excision under certain conditions still has its adherents; it will be discussed further on.

Cauterizations, applications of mercurial, calomel, iodoform, etc., salves should be avoided; clean moist dressings, with glycerolated starch or borated vaseline are sufficient.

Under the influence of energetic general treatment, a chancre will heal in less than ten days.

Secondary Stage.— From thirty-five to sixty days after the appearance of the chancre, generally at the end of forty-five days, the secondary symptoms develop. These are sometimes multiple, profuse and stormy, such cases being described as *secondary explosions*. All possible degrees may occur between the nearly complete absence of all manifestations and the forms described as *precocious malignant syphilis*.

The secondary lesions may affect a large number of organs or organic systems. *Generalized glandular enlargement is never absent.*[1]

Roseola, papular eruptions, mucous patches and headache, are

[1] The generalized adenopathy becomes progressively established in the course of the primary stage. In case of chancre of the genital region, the inguinal bubo appears from the ninth to tenth day after chancre; the cervical, supraclavicular, middle and suboccipital glands become perceptible to touch by the twelfth to fifteenth day; the epitrochlear glands toward the eighteenth day. The value of the presence or absence of generalized glandular enlargement for the diagnosis of syphilis is considerable and too often overlooked; I have emphasized its importance for many years in my hospital service.

extremely common; neuralgias, myalgias, arthralgias, deep bone-pains are frequent; alopecia, iritis, albuminuria, abortion, are not rare.

The general condition is more or less disturbed; there often is an anemic pallor, with rather marked leukocytosis; loss of strength, anorexia, emaciation, neurasthenia, enlargement of the spleen.

In women especially, fever with an irregular course may be observed; a typhoidal state ("typhose syphilitique") has even been described but such cases are probably due to superadded infections. [In exceptional cases the temperature may reach or exceed 104° F. (40° C.)].

These various secondary manifestations may becomes endlessly combined, their coincidence often facilitating the diagnosis. However, in a fair number of cases the cutaneo-mucous eruptions or secondary syphilides which are alone to be described in this book, present themselves separately without other symptoms besides generalized glandular swelling.

Secondary Syphilides.—Their general features are their polymorphism, their abundance, their dissemination, their insidious non-inflammatory development, their painlessness and the complete absence of itching.

The lesions are usually of a rounded form, sometimes arranged in rings, loops or clusters (See Fig. 39 and 127); their color is yellowish-red, like bacon, sometimes coppery, or of a so-called sombre hue. A development in successive crops, a tendency to spontaneous involution and frequent recurrences complete their general properties.

The eruptive lesions of secondary syphilides belong to four principal types: *erythema* (roseola); the *papule;* the *ulceration;* exceptionally, the *bulla* (in congenital syphilis).

These various morphological types may be seen in juxtaposition, constituting polymorphous eruptions.

In a general way, however, one type almost invariably predominates; I have described these eruptive forms in the first part of this book, to which the reader is referred; here it will suffice to retouch the picture.

1. The *erythematous syphilides* are *simple roseola*, which is almost regularly the first eruption; and *circinate roseola*, delayed, recurrent and rebellious (p. 41).

2. The *papular syphilides*, often precocious, include, according to the size of the lesions, a *lenticular* or common form (p. 142); a *miliary* form which is follicular and obstinate (p. 396); a *nummular* form (p. 144); and numerous morphological varieties: papulo-squamous or psoriatiform (p. 113); papulo-crusted (p. 172); and vegetative (p. 247). To the papular type should be annexed, in my opinion, the palmar and plantar *keratodermic syphilides* (p. 216), which may be precocious or delayed, but invariably rebellious.

3. The *ulcerative syphilides* are especially serious and constitute varieties which are malignant from the start (p. 286).

4. The *bullous syphilides* are peculiar to precocious hereditary syphilis (p. 177).

This enumeration does not exhaust the list of cutaneous manifestations of the secondary stage; there still remain to be mentioned:

5. *Pigmentary disturbances* (p. 326).

6. *Lesions of the adnexa*, the hairs (p. 411) and nails (p. 436).

It is important to note that these various types of lesions are characteristic only on the skin. On the mucous membranes and even on the epidermis in macerated regions, these types lose their distinctiveness and tend toward a more or less erosive and oozing, hence highly contagious common form, which is currently designated under the name of *mucous patch*.

Secondary Syphilides of the Mucosa or Mucous Patches.—This is the most common among secondary lesions and it may be stated that few syphilitics are exempt from it. Through their contagiousness, mucous patches acquire social importance, for they are responsible for most transmissions.

They usually appear after the first eruptive manifestation; they often recur repeatedly during the first two or three years; especially when provoked by local irritations—tobacco and a bad condition of the teeth for buccal or pharyngeal patches; uncleanliness for genital and anal patches. They have been noted in the sixth and eighth year and are held responsible for the very rare and extremely doubtful cases of syphilitic infection in the twelfth and even in the eighteenth year.

Mucous patches may be situated at any point of the mouth, the lips, the isthmus and the pharynx, especially on the tonsils and faucial pillars, the commissure of the lips and in the vicinity of carious teeth; at any point of the vulva; on the prepuce and glans, around the anus, on the conjunctivæ, in the nasal fossæ, in the larynx; sometimes even in the axillæ, at the umbilicus, or more frequently between the toes. The syphilides designated as *mucous patches of the skin* have been previously mentioned (p. 144).

A distinction is made between several varieties of syphilides of the mucous membranes, which correspond to the cutaneous syphilides, representing a possible localization of the latter.

Erythematous syphilides, simple red spots, are seen especially on the palate, on the cheeks and on the labia minora.

Opaline plaques, ordinary mucous patches, the most widely distributed variety, are characterized by a slight, sometimes hardly papular elevation, on which the epidermis is swollen and whitish, not eroded nor proliferating. They are encountered everywhere, notably on the velum of the palate or at the vulva; they may assume a fairly characteristic *circinate* arrangement, in arcades.

The pharyngeal and buccal mucous patches are sometimes *diphtheroid,* to such a degree that a confusion with diphtheria occurred in cases reported by Fournier. It is probable that this appearance is the result of a superadded infection (fuso-spirillary association, etc.).

Erosive or *papulo-erosive syphilides* are papular syphilides whose squamous epithelium has become detached; they present the appearance of distinctly rounded red erosions; they often become, or have been opaline. The papular mucous patches are known outside of France as *flat condylomata* [condyloma latum].

Papulo-hypertrophic and *proliferative mucous patches* develop especially in uncleanly persons, at the vulva or in its vicinity, in the genitocrural and intergluteal folds and sometimes at the buccal commissures; on the tongue, notably on its posterior third, they may constitute what the French call "toad's back tongue." I have seen enormous patches of this kind on a wholly neglected scalp. They consist of elevations the size of a pea to that of the thumb or larger, sometimes nearly a centimeter thick, with a granular or papillomatous surface, discharging a turbid and yellowish serous fluid with a fetid odor.

Ulcerative syphilides of the mucous membranes are less common, they are observed on the lips, at the commissures, the gums, the pharynx and on all parts of the vulva.

Certain syphilitic lesions of the tongue known as *smooth patches* or "mowed-lawn" patches are usually regarded as related to mucous patches. These are dry, red, non-eroded patches deprived of their papillæ, usually of an oval shape; they may become papular. Their onset is often delayed and may even occur in the course of the tertiary stage; Fournier considered them as contagious. They are very slightly amenable to mercurial treatment and last several weeks or even months.

Although usually painless, mucous patches may interfere with movements, causing dysphagia, etc.

The *diagnosis* of mucous syphilides is easy when they occur in the course of a long train of symptoms; inversely, it frequently happens in practice that they furnish valuable information in doubtful cases; but it would be venturesome to base a diagnosis of syphilis exclusively upon the presence of mucous patches, even when they assume an appearance which may seem to be typical.

The principal causes of error are as follows, in the mouth: aphthæ are entirely round, yellowish with a narrow carmine margin and very painful. Herpes appears suddenly, is painful and polycyclic. The bullous dermatoses, hydroa, may produce lesions almost identical with mucous patches. *Perlèche* is an opaline erosion situated exclusively at the commissures of the lips, but encroaching upon the cutaneous

epidermis of the vicinity; it is epidemic in schools and due to a streptococcus. Glossitis arcata exfoliativa is characterized by its special border. Leukoplakia, buccal lichen planus and median rhomboidal glossitis, etc., are dry, stationary or very persistent lesions. Mercurial stomatitis is a diffuse inflammation, sometimes with peridental or lingual ulcerations closely resembling mucous patches; a coincidence is possible.

Sources of error in the genital region are represented by: traumatic lesions, which are irregular and ephemeral; erosive balanoposthitis, which is characterized by whitish circinate forms which may be removed by friction.

Soft chancre is purely ulcerative and suppurates profusely; it is auto-inoculable. Proliferative herpes of the vulva, a very rare disease, closely simulates the papulo-erosive syphilides. The same is true for the post-erosive papular erythema of nursing infants (Fig. 1). Finally, in uncleanly individuals, one may see in the vicinity of the anus absolutely syphiloid patches of proliferative dermatitis, which are of commonplace character (A. Fournier and Brouardel).

[The demonstration of *spirocheta pallida* on the surface of a lesion of the mucosa is not always easy. When it can be made with certainty the diagnosis is placed beyond a doubt.]

Tertiary Stage.—While it is possible to enumerate in a few lines the principal secondary syphilitic lesions and to give an idea of the course of this stage, such a sketch is altogether impracticable in regard to tertiary syphilis; its domain is too extensive, its course too varied and too irregular. I shall accordingly discuss only its cutaneous and mucous lesions.

Tertiary Syphilides.—The general features distinguishing these from the secondary symptoms are as follows: They are deep and serious, not superficial and benign lesions, of an ulcerative or sclerotic type, not undergoing absorption; limited and regional, not profuse and scattered; monomorphous, often with a well-marked tendency to become arranged in groups and to assume a circinate configuration, "to become disciplined" to use an expression of Fournier's.

The circinate or rather semicircular arrangement of an eruption would be incorrectly interpreted as characteristic of syphilitic lesions; in the first place, its constituent eruptive lesions must be taken into consideration. The following general statements can be made: While rare in secondary syphilis (syphilides in arcades, mucous patches in arcades), a circinate arrangement is very common in the tertiary syphilides. It is encountered furthermore in some cases of lupus, in some eczemas and psoriasis, in many erythemas, in recurrent pemphigus, sometimes in mycosis fungoides, in lichen planus, lichen scrofulosorum, parapsoriasis en plaques, etc. The

eczematides and more particularly the cutaneous trichophytosis, rather form complete circles.

The question of the contagiousness of these tertiary lesions had been settled in the negative sense or very nearly so. Modern methods of investigation, however, notably inoculation into monkeys, have shown that a certain number of these lesions are virulent, the proportion being difficult to indicate. We do not know if the spirochete, as has been supposed, exists here in a different form from that which it assumes in chancre and in the secondary manifestations [but there is no necessity for this assumption; a gumma is the effect of a spirochete *in loco* acting on a soil modified by the existing disease. Spirochetes have been found in gummas though in very small numbers. I am of the opinion that encysted or resting forms must also exist; but this lacks proof.]

The morphological aspects under which the tertiary syphilides may manifest themselves have been previously described; they are classified under six headings, as follows:

1. *Tertiary erythemas* (p. 41).
2. *Tubercular syphilides,* circinate or in patches (p. 253).
3. *Ulcerative syphilides,* of several varieties (p. 285); tuberculo-ulcerative, atypical gummous, sclerogummous and phagedenic.
4. *Syphilitic gummas* (p. 267).
5. *Tertiary vegetative syphilides* (p. 247).
6. *Diffuse hypertrophic syphilomas* and *syphilitic elephantiasis* (p. 368).

These various lesions may occupy any region of the integument or of the mucous membranes. It may serve as a useful guide, however, to mention the types which occur especially in some special regions.

The center of the face, the nose and the lips, is one of the elective foci of tertiary syphilis in all its forms, dry tubercular, ulcerative, proliferating and leontiastic. Superficial confluent tubercular syphilides, closely analogous to rosacea, are also observed in this location.

In the mouth, on the palate and the pharynx, superficial tubercular and ulcerat ve syphilides are met with, as well as perforating gumma.

On the tongue, there occur gummas, gummous and sclerogummous infiltrations, sclerotic glossitis and sometimes tubercles.

On the scalp, gummas and tuberculo-ulcerative syphilides are not uncommon; the crusted and superficial variety of the latter may simulate psoriatiform eczematides. To the palmar and plantar regions belong the psoriatiform so-called keratodermic syphilides. On the extremities, syphilitic elephantiasis may develop. On the external genital organs of both sexes, gummous infiltrations and gummas are found, as well as proliferative, chancriform, phagedenic syphilides, etc.

For the diagnosis of each of these forms, the reader is referred to the description of the eruptive lesion which distinguishes it (Part I, Morphology).

Congenital Syphilis.—The teachings on the subject of congenital syphilis and the multiple problems involved in its investigation have been greatly influenced by recent scientific acquisitions, the discovery of the spirochete and the introduction of the Wassermann test. Without entering into details, I shall limit myself to stating the conclusions which are agreed upon or at least tend to become generally accepted.

Hereditary syphilis, better named congenital, is always of maternal origin; it is transmitted from the mother to the fetus by way of the placenta rather than through the germ-plasm or the ovum.

As the mother of a child born with syphilis is always herself syphilitic, even if she appears healthy (Wassermann reaction), this fact affords a simple explanation of Colles' law (immunity of the mother of a child born with syphilis) and Profeta's law (immunity of an apparently healthy child toward its syphilitic mother). So-called conceptional syphilis, or through reflex ("choc en retour") *in utero* (syphilization of the mother by a fetus conceived from a syphilitic father) is an error in interpretation of the facts. A syphilitic father can transmit virulent syphilis to his child only through intermediate infection of the mother; but on the other hand, without syphilizing the mother he can transmit to his offspring, like an alcoholic or tuberculous father, both physical and psychic disturbances of development and a tendency to malformations; this heredo-dystrophy may or may not be associated with congenital syphilis of the child.

Congenital syphilis differs from acquired syphilis in a fundamental factor: the infection, of placental origin, takes place by the hematogenous route and is visceral from the start. The primary stage is accordingly absent in hereditary syphilis; there is no chancre; moreover, the secondary symptoms and the visceral manifestations of tertiary character are intermingled instead of successive. The course is therefore much less regular.

In addition, the specific manifestations may become associated with dystrophic disturbances, malformations and various pathological tendencies, due to poisoning of the germinal tissue, spermatozoa and ova, as well as infection of the entire maternal organism, so that it is readily understood that the clinical picture of congenital syphilis may be rather polymorphous.

[The offspring of a woman in the early stages of syphilis is always syphilitic; in the later stages the child may or may not be syphilitic. In a series of pregnancies the first products of gestation may be abortions or dead children, then children born alive and syphilitic,

then a series of apparently healthy children. Some of these apparently healthy children will really be free from syphilis and some may develop signs of syphilis within a few years; or possibly the first lesion will appear only after the lapse of many years. Sometimes among the later children of a series, healthy and syphilitic children alternate.

The explanation of these phenomena is simple but not generally understood. The blood of the syphilitic mother in the first year or two after infection is loaded with spirochetes some of which are sure to enter the uterine arteries and infect the fetus, producing there the active lesions of syphilis. In the tertiary stage of the mother's syphilis the spirochetes have become fixed in her tissues and her blood-stream only occasionally contains a few spirochetes. Under these conditions it is a matter of chance whether or not the spirochetes will reach the placenta and thus infect the fetus. But the blood of the fetus shares the quality of its mother's blood; in late syphilis, the fetal blood is *ab ovo* the blood of a tertiary syphilitic and contains the same substances which have given to the mother that kind of immunity which Neisser called anergy. It is this condition which renders her immune to a fresh infection though it offers no protection against local tertiary lesions. The spirochetes of a tertiary syphilitic mother that happen to penetrate to the fetus find conditions there like those in the mother. They produce no acute syphilis but becoming localized in the tissues develop the late lesions of syphilis hereditaria tarda.]

The cutaneous and mucous manifestations alone are to be considered in this book; the other symptoms will accordingly receive merely a brief mention.

A classical distinction is made between early and delayed manifestations of congenital syphilis.

Precocious congenital syphilis sometimes manifests itself at birth by a peculiar *facies:* the skin is lifeless, wrinkled, flaccid, withered, like that of a very old man; the skull is deformed, with an abnormally high forehead ("olympian brow"), a natiform cranium, hydrocephalus, dilatation of the cranial veins and general athrepsia (Fig. 182).

At birth, children with congenital syphilis may present three symptoms: *syphilitic pemphigus;* very frequently, an irritative serous, later purulent, *coryza,* drying in greenish crusts and interfering with respiration and nursing; *mucous patches,* situated especially on the lips, often at the commissures, either fissured or radiating, rose-colored or opaline, oozing, crusted, sometimes with an indurated base and painful; they are a common cause of infection of wet-nurses.

Very soon, in the first weeks of life and usually before the fourth

month, the *cutaneous syphilides* make their appearance. These have been especially well studied by Jacquet.

Polymorphous *erythemato-papular* syphilides consist of more or less rounded salmon-colored or dark-red erythematous spots, situated on the buttocks, on the lower limbs, on the neck, around the mouth or in the folds. Some fade and disappear while others increase in size and become scaly, or thicken into papular discs; the elevation is often marked at the borders (*circinate erythemato-papular syphilide*) and sometimes at the center of the spots (*extended papulo-lenticular syphilide*). A well-marked "collarette of Biett" (p. 142) may be observed at their circumference.

Jacquet has pointed out that the palmar and plantar syphilides, known as syphilitic pemphigus, are of the same type and become bullous only in consequence of local factors.

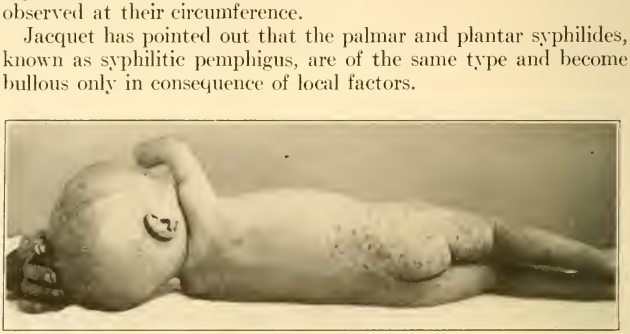


FIG. 182.—Early congenital syphilis; child of three months. Note the hydrocephalus, the dilatation of the cranial veins, the papulo-erosive syphilides of the buttocks, loins, thighs, etc., the large abdomen and the puny limbs.

The erythemato-papular syphilides may become *erosive* through maceration, or *crusted* and *impetiginous*, or sometimes *ulcerative* at points exposed to pressure or irritated by fecal matter; or, rarely, very scaly and *psoriatiform*. Furthermore, an *acneiform* syphilide has been described, formed by small umbilicated papules, centered by a small crust and agglomerated in patches.

These polymorphous erythemato-papular syphilides are often very difficult to distinguish from the gluteal erythema of the newborn (p. 31). They are the substitute in congenital syphilis of the roseola and the various papular syphilides of adults; as a rule they are associated with mucous patches and sometimes with lesions of the nails and alopecia. Gummas are rare.

Proper *treatment* of newborn infants having congenital syphilis saves the lives of the majority [without proper treatment very few of them survive even a year]. These children must never be entrusted to a healthy wet-nurse, but must be nursed by their own mother. In order to protect their digestive functions, mercurial

medication by the mouth with van Swieten's fluid, etc., is preferably replaced by mercurial inunctions (1 or 2 grams of mercurial ointment daily). It is advantageous to administer injections of novarsenobenzol in watery solution (5 mg. as the maximum dose per kilogram of body weight per week) into a scalp vein or in oily suspension into the buttock.

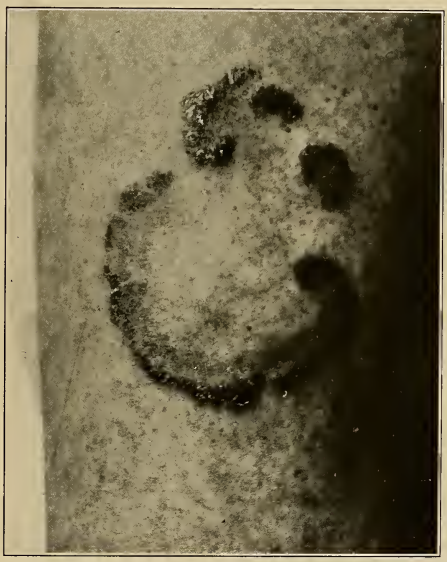


Fig. 183.—Congenital syphilis. Circinate tuberculo-squamous syphilide on the thigh of a girl aged eleven years.

Syphilis congenita tarda is the form observed in older children, in adults or even in old persons. For this chapter of pathology we are almost entirely indebted to the contributions of my teacher, A. Fournier, prior to whom the remote evidences of congenital syphilis were generally referred to scrofulo-tuberculosis, rickets, etc. It has been claimed that these patients must have had early manifestations and that it is only the diagnosis that is delayed in such cases. This is probable but not certain. The didactic and practical importance of the subject is nevertheless considerable.

The remote symptoms of congenital syphilis are in part specific and in part dystrophic in character.

Decidedly specific are those which plainly present the features of tertiarism (Fig. 183); these require therefore no special description. Aside from the external integument, they affect chiefly the velum of the palate, the pharynx, the tongue, the nasal fossæ and the genital organs and are as a rule essentially ulcerative and destructive.

Under the name of *stigmata of congenital syphilis*, a series of malformations, developmental disturbances and lesions have been grouped, whose characters point to syphilis in the parents on simple examination of the patient. Some are of a dystrophic character, while others are residues of specific lesions; efforts are now under way to separate these two groups of symptoms.

The principal signs are the following: Hutchinson's triad, namely dental malformations (Fig. 184), ocular lesions and auditory disturbances; bony deformities such as cranial and nasal malformations, "sabre-blade" tibia and exostoses; testicular atrophy; infantilism; gluteal cicatrices of Parrot and radiating cicatrices of the lips.

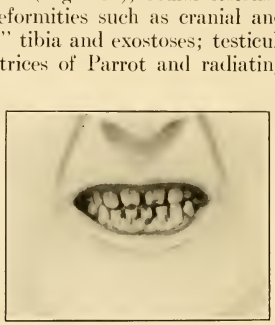


Fig. 184.—Congenital syphilis. Hutchinson's teeth.

Pathological Anatomy.—Before the discovery of the spirochete, the syphilitic lesions had already been recognized as possessing the features of an infectious inflammation; this affects in the first place the arterial and venous bloodvessels, altering the vascular walls and giving rise around them to the production of cellular infiltrations composed for the most part of plasmocytes.

The more or less circumscribed or diffuse newformations which make up these infiltrations are capable of resolution in the primary or secondary stage of the disease, in the sense that they may undergo retrogression and practically complete absorption; the tertiary newformations, on the contrary, are incapable of resolution and tend to form sclerotic tissue, provided they do not undergo necrobiosis; this may occur early, while the pathological tissue still has an embryonic structure, or later, after the sclerosis has become established. The name of gummous degeneration is employed to designate this necrotic process in either case.

In syphilitic chancre, the cellular infiltration of the cutis is very dense; the connective-tissue bundles are swollen, the walls of the bloodvessels are greatly inflamed, the epidermis is thickened at the borders, but has lost a number of its layers at the center, the remainder being infiltrated with leukocytes and fibrine as well as degenerated; in many cases, thinned and elongated interpapillary buds are its only residue. The treponema flourishes, especially in the vascular walls, whence they seem to emigrate in part into the vascular lumen, which is very important in view of the generalization of the infection; in part, into the connective-tissue bundles and spaces, whence the lymphatics carry them into the glands; and finally in the epidermis, where they may be transmitted by contagion.

The lenticular papule may serve as the model of a secondary syphiloma (Fig. 31). The infiltration of plasma cells interspersed with a few giant cells is arranged in more or less continuous perivascular cuffs. The epidermis is merely stretched; or it may be parakeratotic, or edematous and infiltrated with leukocytes, or degenerated, resulting in the formation of scales, crusts, erosions or ulcerations.

In the *roseola* the cellular infiltration is very slight. It is somewhat more considerable in the mucous patches, which may have the structure of papules, with an opaline epithelium through separation of the cells and very abundant immigration of white corpuscles. These lesions are literally teeming with spirochetes.

The tertiary lesions differ from the preceding in their course rather than in their characters; the treponema, however, has only very rarely been demonstrated.

Genuine syphilitic gumma seems to originate through thrombophlebitis in the vascular plexus of the hypoderm; the profuse cellular newformation of which it consists undergoes liquefaction while at the same time the stroma becomes necrotic.

In syphilitic tubercle, the newformation, in which the bloodvessels and lymphatics are greatly dilated, is partitioned by sclerotic strands. As compared with the papule, it is therefore more fibrous, hence its hardness; contains more venous blood, hence its dark color; and the sclerosis persists after its disappearance, hence the cicatrix which it leaves behind (Fig. 87). The epidermis may be hypertrophied in the proliferative varieties; or it may be edematous, degenerated and infiltrated with leukocytes in the tuberculo-crusted and tuberculo-ulcerative forms. The crust, which characterizes the last two forms as well as the secondary ulcerative syphilides of the precocious malignant form, develops at the expense of the epidermis and the infiltrated necrotic upper layer of the cutis; it accordingly precedes the ulceration. Endarteritis and phlebitis are regularly

found around all tertiary syphilomas. All these forms may present a tuberculoid structure (Nicolas and Favre).

The lesions of congenital syphilis do not markedly differ from those of acquired syphilis. The abundance of spirochetes in its early manifestations has already been referred to.

Diagnosis.—The diagnosis of syphilis should always be guarded; I repeat that the patient should be very cautiously informed of its existence.

Sometimes the diagnosis is obvious from the start, while in other cases, numerous and painstaking investigations are required. It may happen that in spite of these investigations no definite conclusion is possible. Even in countries supplied with well-informed physicians, a considerable number of previously undiscovered cases of syphilis are met with and this number has grown since our knowledge of the specific origin of the so-called parasyphilitic diseases and since the common employment of the Wassermann reaction.

In connection with every type of syphilitic symptoms, I have indicated its diagnostic features, based on its objective characteristics. Some of these are indicative, notably the typical hard chancre, roseola, lenticular papular eruption, arciform syphilides, alopecia areolata, pigmentary syphilide of the neck, circinate tertiary syphilides, etc.

When it is not possible to affirm the existence of syphilis from the morphological aspect of a given lesion, as is true in by far the majority of cases, there is a series of indirect measures at our command that enable us to arrive at a certainty; these are of unequal intrinsic value and not indiscriminately applicable in all stages of the disease; nor is it always possible or necessary to employ them all.

Among these procedures, some are of a *clinical* nature and furnish only more or less probable data.

They are based upon: (1) the presence of concomitant lesions; certain associations are almost pathognomonic; I have emphasized the value of progressive general glandular enlargement; (2) the regular succession and sequence of the lesions; and (3) on the history of exposure to contagion and the examination of the contaminating party (Bassereau). The ex-juvantibus method [test by treatment] has too many serious disadvantages to justify its employment.

Recent discoveries have enriched these traditional diagnostic principles by the introduction of new scientific procedures belonging to the domain of the laboratory.

The demonstration of the spirochete, in stained smears or by direct ultra-microscopical examination of the serous discharge, constitutes the method of choice in case of oozing lesions of the

first stages, namely a chancre or mucous patches; a positive result is absolute proof. However, this demonstration fails in the ulcerative lesions, even of the secondary period; it is very complicated in non-ulcerative syphilides and negative tests, even when repeated, possess no value. In the case of tertiary syphilides, this method is of no assistance at all.

The demonstration of the spirochetes in biopsy-sections requires more time and a more elaborate technic; its value is open to the same objections as those of the direct procedure. It is applicable to the non-ulcerative lesions of the most virulent stage of the disease.

The method of demonstrating the virulence of a lesion by inoculation into monkeys, as formulated by Thibierge and Ravaut, or by inoculation of the rabbit's cornea or scrotum, is absolutely conclusive when the outcome is positive. But its inherent difficulties and the delay prevent its employment as a customary procedure.

The *sero-diagnostic* method of Wassermann-Bruck-Neisser, based on the complement-fixation method of Bordet-Gengou, depends, not upon the demonstration of the noxious agent, but upon the demonstration of the resulting humoral changes. Its technic is extremely delicate; it necessitates minute precautions with controls and a correct interpretation of the findings.

The fact is now known that the Wassermann reaction is not strictly specific, for a positive reaction is yielded by yaws, trypanosomiases and leprosy. In a general way, however, under ordinary conditions and with a reliable worker, a positive outcome may be credited with absolute value and a series of negative results with a value of great probability.

Sero-diagnosis is not applicable in the initial stage of the chancre, because the reaction does not appear until about three weeks after the onset of the primary lesion. It is a valuable aid in the secondary stage and especially in the tertiary stage. It is well to keep in mind, however, that the only information supplied is to the effect that the individual is syphilitic, without justifying a statement that a given doubtful lesion presented by this individual is itself of syphilitic nature. The Wassermann reaction is most serviceable in the latent stages of the disease, the other methods, except the luetin test which will be discussed presently, being inapplicable at these times. It may also be utilized to a certain extent in the direction of the treatment.

Milian and others have studied under the name of *reactivation* the reappearance of a positive Wassermann reaction under the influence of treatment with arsenicals; it is claimed to occur early (from the fifth to eleventh day) in the secondary stage, but is transitory; rather delayed (toward the fifteenth day) and generally prolonged in the tertiary stage. [In my opinion the "provocative"

Wassermann test is not only useless but is apt to be misleading. Cf. *Am. Journ. Syphilis*, April, 1919.]

For the *cutaneous reaction* to *luetin*, credit is due to Noguchi (1912). Luetin is a sterilized emulsion of a culture of six strains of Spirochæte pallida; it is employed in intradermic injections. The results yielded by it are not always very reliable. A positive reaction manifests itself after twenty-four to forty-eight hours, by the appearance of a papular or pustular elevation which lasts a week or longer; sometimes it is sluggish or delayed.

A negative reaction is erythematous or faintly papular and subsides by the fifth day. It is claimed that the percentage of positive results is only 33 in the primary stage and 47 in the secondary stage, but from 80 to 90 in tertiary syphilis, whether latent or congenital; so that there is no conformity with the Wassermann reaction. A certain number of non-syphilitic persons react likewise. An analogous reaction to that of luetin, which is on the whole only a manifestation of allergy has been obtained on the other hand with extracts of syphilitic organs and even with other substances. The value of the luetin-reaction is still a matter of controversy. [That the reaction is not specific is shown by the fact that it may occur after an injection of agar (Stokes) and possibly other substances; moreover a reaction is regularly obtained in non-syphilitics who have ingested iodides as long as a month before.]

The *histological diagnosis* of syphilis through examination by biopsy has been relegated to a secondary rank; under certain conditions and especially in combination with sero-diagnosis, this method may prove serviceable.

It will be readily understood that the various procedures outlined above may be combined in several ways according to the supposed stage of the disease.

Treatment.—Syphilis is one of the diseases against which we are in a good state of preparedness.

Lacking a preventive or curative serum, which remains to be discovered, we possess mercury which often does wonders when it is properly employed and well tolerated; and there are furthermore the recently introduced arsenical compounds, which are still more powerful; a few medicinal adjuvants and hygienic prescriptions complete our therapeutic armamentarium.

Mercury.—It is not known if mercury acts directly as a parasiticide, or through the intermediation of antibodies, or by increasing the defensive reactions of the organism, etc. A few days of treatment suffice to render the demonstration of spirochetes in the syphilitic lesions impossible. At any rate, mercury not only cures the symptoms but also prevents relapses and tertiarism and even hereditary transmission. Like many others, I have been able to

note that the vast majority of syphilitics who were treated with mercury alone for a long time and with good doses remained definitely free and acquired a permanently negative sero-reaction.

The mode of introduction of the medicinal agent is not of capital importance in itself, the essential factor being the amount which has really been absorbed. The reason why the old mercurial fumigations have been abandoned and the obstacle to the adoption of the inhalation method, through the mercurial flannels of Merget or through the powder- or ointment-bags of Welander, which the patient must wear on his person night and day, consist in the uncertainty as to the dosage absorbed under these methods of administration.

Mercurial inunctions are open to the same criticism; furthermore, they are dirty and suggestive. They enjoy great popular favor, however, especially abroad; for children of tender years, they constitute the procedure of choice. [A 30 per cent. calomel ointment seems to be not less efficient, and has the avantage of cleanliness.]

The full-strength or 50 per cent. mercurial ointment is employed for these inunctions, in daily doses of 6 or 7 grams for a man, 5 grams for a woman, 1 or 2 grams for a newborn child. The inunctions must be applied for a long time, during ten to fifteen minutes, until there is apparent penetration; the regions should be carefully alternated, rubbing on the first two days one of the sides of the thorax, on the following days the internal aspect of one of the limbs, after which the series is resumed, avoiding the axillæ, the groins and the scrotum, which are too much exposed to hydrargyria. The treated part is covered with gauze for the night; on the following morning it is carefully cleansed with soap and dusted with powder. [Since mercury is slowly vaporized at the body temperature a part of the effect of inunctions is due to respiratory absorption. Inunctions are therefore most effective when made in a small room with closed windows.]

The buccal route, for a long time the most generally used, also has its disadvantages: uncertainty as to the actual penetration of the dose into the tissue-juices, low effectiveness and lasting digestive disturbances; the latter attract little attention on acount of their delayed appearance, but occur almost infallibly in patients who have been treated for a long time.

Use is made either of van Swieten's fluid, 4 to 6 teaspoonfuls daily in a little milk, or Dupuytren's pills (bichloride of mercury and extract of opium āā 00.1, excipient and glycerine q. s. for 1 soft pill) or especially Ricord's pills (protoiodide of mercury 0.05, extract of opium 0.01, excipient and glycerine q. s.) to be taken at meals, in a dose of 2 pills daily for an adult, 1½ for a woman.

The rectal route may be utilized. Audry recommended supposi-

tories of cacao butter with an addition of 40 per cent. gray oil, in such a proportion as to contain 0.02 to 0.04 of metallic mercury; this method is slow in its action.

Mercurial injections are at present one of the fundamental methods of treatment of syphilis. They insure a strict dosage, exclude fraud or negligence on the part of the patient, spare the digestive apparatus and when properly applied and well tolerated are found to be almost as rapidly effective as arsenical medication. At the present writing, I regard mercurial injections as an indispensable complement of treatment with the arsenobenzols. Further on I shall point out how the two medications may be combined.

Mercurial injections are made in the region of the buttocks. The pain of the puncture is practically zero, abscesses are no longer to be feared, pain coming on after the injection is insignificant provided the technic is correct and nodosities are rare and usually painless. On the whole, the drawbacks of the method cannot be compared with its advantages.

The technic is now definitely established and the following rules must be observed: The necessary implements are a needle from 6 to 7 cm. long, preferably of iridium-platinum and an accurately divided sterilizable syringe made entirely of glass; the whole outfit is first to be boiled. The puncture-points are selected as follows: (1) On a horizontal line passing three or four fingers' width below the iliac crest; (2) on a vertical line passing two fingers' width from the intergluteal fold. It is advisable to begin with the left buttock and to inject on the two sides alternately, of course avoiding the previously punctured points. The patient lies on the abdomen, with complete muscular relaxation; the area selected is cleaned with a cotton wipe soaked in benzine, ether or alcohol. The needle is plunged in vertically with a clean thrust and a minute is allowed to pass, in order to be sure that no drop of blood flows through its lumen; should this happen, the needle will have to be [partly] withdrawn and inserted elsewhere, to guard against embolism; otherwise, the syringe containing the desired dose is adjusted [and the fluid slowly injected]; at the end of the procedure, a drop of collodion or a piece of plaster is placed over the puncture-orifice.

Mercurial injections are administered with insoluble compounds or with soluble salts.

The former have the advantage of being applicable at longer intervals, usually once a week. The soluble injections must be repeated every day, causing a loss of time and much inconvenience outside of hospital practice; they have a powerful and rapid, but less durable effect.

Gray oil is the most practical insoluble injection. The formula of the Codex for 1908 contains: purified mercury, 40 grams; lano-

line, 26 grams; vaseline oil, 60 grams; 1 c.c. contains 0.4 gm. of mercury. This preparation is employed luke-warm, using a syringe of narrow caliber [tuberculin syringe]; 0.07 to 0.12 Hg weekly should be injected into a man and 0.06 to 0.10 into a woman.

Calomel injections are much more energetic and efficient; their action is nearly as rapid as that of arsenobenzol. They are often alone sufficient to cure certain lesions, such as the obstinate palmar syphilides and the tertiary lesions of the tongue. The dose to be injected is from 0.05 to 0.10 (or 1 c.c. of the formula: calomel (by vaporization) 0.5 or 1.0, vaselin and vaselin oil, āā 5 grams). Their drawback consists in the sometimes severe but as a rule quite tolerable pain caused by them. This is largely obviated, as I have suggested, by adding per 1 c.c., 0.03 of guaiacol and 0.02 of camphor. The same addition is advantageously made to gray oil and to the other insoluble injections.

The basic salicylate of mercury (0.10 per injection), or yellow oxide (0.05 to 0.10) may be substituted for gray oil, without, however, being preferable to it.

[With all the mercurial suspensions in oil, it would seem physiologically preferable to use olein (ol. olivarum) rather than foreign bodies like the paraffins (vaseline, vaseline oil, etc.). Furthermore, the larger the volume of the injected mass the greater the pain. I employ calomel in 20 per cent. suspension and the salicylate in 33 per cent. suspension, injecting a proportionately smaller amount of fluid.]

Soluble injections are made with benzoate of mercury according to the formula of Gaucher; with bibromide or biniodide of mercury, or with the cyanide. The daily dose is from 0.01 to 0.03 of each of these salts; the pain is generally moderate. Bichloride of mercury is more painful and should be rejected for this reason. Tolerance varies greatly according to the patients and it is advisable to look in each particular case for the most readily tolerated formula. Various preparations of mercury disguised under proprietary names (hermophenyl, enesol, etc.), are not sufficiently reliable in their action.

Intravenous injections of soluble salts may also be recommended; the cyanide of mercury alone is used for the same [the bichloride is also employed]. They are entirely painless and very promptly efficient. [They are administered in normal saline solution.]

Local mercurial treatment of syphilides, especially the tertiary manifestations, is entirely reasonable; it serves as an adjuvant of the general medicinal treatment and prior to the era of arsenobenzol injections was recommended by me in those cases where the patient's buccal or intestinal condition prevented an energetic general mercurialization. I have been well pleased, especially in the treatment of

circumscribed leukoplakias and leukoplasic ulcer, with local injections of a few drops of an isotonic cyanide solution of 1 to 3000 (formula: cyanide of mercury, 0.33; cocain chlorhydrate, 5.0; sodium chloride, 7.0; sterilized water, 1 liter) repeated twice weekly. The traditional mercurial plaster, salves and mercurial baths are no longer indicated at the present time.

Mercurial complications, hydrargyria are possible with all compounds, all formulas and all methods without exception. Their possible occurrence renders it necessary first to test the patient's sensibility and always to proceed with caution.

Mercurial stomatitis was a distressing and formidable symptom in the days when salivation was systematically aimed at. The present tendency is to consider it as due to the fuso-spirillary association and induced by the irritation of the mouth. Its development may be prevented by putting the mouth in good condition before beginning the treatment and by maintaining great cleanliness. The use of tobacco must be interdicted. The gastro-intestinal apparatus likewise requires continuous supervision. Finally, the urine must be watched.

Arsenic.—Arsenic in all its forms had long been advocated as an adjuvant of mercurial treatment; sodium arsenate, the cacodylates, methylarsenate, hectargyrium and other preparations have served for this purpose.

Since a dozen years, however, a direct specific medication has been sought in new arsenical compounds. Atoxyl and arsacetine were promptly abandoned as too dangerous. The "hectine" of Mouneyrat has enjoyed a certain popularity, but this product does not seem to me to be better than a good adjuvant.

In the course of his investigations on the arsenic treatment of the spirilloses, Ehrlich in 1909 stopped at a compound, the dichlorhydrate of dioxydiamido-arsenobenzol, which was numbered 606; this product was labelled with the name of salvarsan; at the present writing it is manufactured everywhere and known as *arsenobenzol*. [In the United States the official name since 1917 is *arsphenamine*.] Shortly afterward he recommended a related product, much more convenient to employ, the dioxydiamido-arsenobenzol-monomethylene-sulfoxylate of sodium, or 914, or neosalvarsan, which is customarily designated by the name of novarsenobenzol [or neo-arsphenamine]. Other more or less analogous arsenical salts, notably galyl, luargol, etc., have been produced and tried in France; these investigations are still under way.

Briefly, arsenobenzol and novarsenobenzol constitute the anti-syphilitic arsenical medication of the present day.[1]

[1] Wherever treatment with arsenobenzol, or the arsenobenzols, has been referred to in the course of this book, I have meant intravenous injections with arsenobenzol or with novarsenobenzol [arsphenamine or neo-arsphenamine].

The hope which had been aroused of a "therapia sterilisans magna," a radical and rapid cure of syphilis has not been realized; it must be abandoned and doses employed which do not expose to the gravest risks. Nevertheless, the efficacy of arsenobenzol and almost to the same degree of novarsenobenzol is not less remarkable; in a few days they will cicatrize a chancre, obliterate roseola or mucous patches; when employed in the primary stage and especially in combination with mercurial injections, they almost certainly protect against all secondary manifestations and usually maintain a permanently negative Wassermann reaction. The test of time is not yet sufficient to state that patients treated in this way will remain free from tertiary and parasyphilitic lesions. While this may be hoped for, it is advisable to complete the treatment with mercury for the present. At any rate, the dangers of contagion are immeasurably diminished in these "silenced" cases of syphilis and it is needless to dwell upon the social importance of this result.

The manipulation of the new arsenical compounds is difficult; their employment cannot be said to be absolutely devoid of danger; especially in the early days, serious and even fatal results have been noted. Better regulated doses and extreme precautions, however, have reduced the risks of this treatment to a minimum.

There is a practically general agreement as to their administration only by the venous route. Those who have witnessed the intolerable pains and fearful sloughs which were often produced by intramuscular injections of 606, such as were originally administered, have abandoned them altogether. Balzer and Dumouthiers advocated intramuscular injections of novarsenobenzol in doses of 0.25, twice daily. These are often not particularly painful, have a slower action and are only rarely indicated. Nowadays, every physician should be familiar with the technique of intravenous injections.

Arsenobenzol is the most active and the most permanently sterilizing antisyphilitic remedy known. Its watery solution is acid and must be neutralized or rendered slightly alkaline by the addition of a sodic hydrate solution, then diluted with physiological salt solution of 7 per 1000, prepared with pure sodium chloride dissolved in freshly distilled water; the total amount to be injected is according to some writers from 150 to 200 c.c., according to others 50 c.c. for each 0.10 of the remedy.

Nov-arsenobenzol is generally preferred on account of the relatively simple instrumentation and mode of preparation of the solution, especially when the very convenient procedure of intravenous injections in concentrated solution of Ravaut is adopted and carried out as follows: Immediately before it is to be used the contents of a nov-arsenobenzol ampoule is dissolved in 5 to 10 c.c. of physiological

salt solution, 4 per 1000, or the same quantity of distilled water or even freshly boiled and cooled water may be employed. Solution is instantaneous; the solution is drawn into an all-glass sterilized syringe, provided with a short-bevelled platinum needle; the patient lies down and the arm is put on the stretch; the needle is now inserted into a vein at the bend of the elbow which has been made prominent by applying a rubber band on the arm; when the blood flows back into the syringe indicating that the needle is properly inserted, the band is removed from the arm and the piston is gently pushed in; after the injection has been made and the needle is withdrawn, no dressing is required. [The field of operation must, of course, have been sterilized with alcohol or a weak solution of iodine.]

Some precautions are needed with all arsenical compounds. In case of virulent syphilis, with multiple manifestations, it is advisable to treat the patient for two or three days before the arsenical injection, with injections of a soluble mercury salt; this guards against an attack of fever with malaise, known under the name of Herxheimer's reaction and attributed to the sudden absorption of the products resulting from a large number of disintegrated spirochetes. The patient must fast three hours before and five hours after the injection. Examination of the urine just before the injection is indispensable, so as to permit a diminution of the dose or its postponement to a later date, in case albumin is present. The ampoule must be carefully examined to see that its contents consist of a light yellow powder, unaltered by penetration of air.

As a rule no reaction of any kind follows. It is advisable, however, to warn the patient of the possible onset of certain symptoms, such as headache, mild diarrhea, a chill with a slight ephemeral fever, general prostration, nausea, sometimes vomiting, rarely an erythematous eruption; he will be so much the more pleased should these symptoms fail to make their appearance.

Threatening, grave and even fatal complications, the mechanism of which has been extensively discussed, have been observed especially with arsenobenzol, more particularly at the time of the first ventures; they have become extremely rare since the doses are more carefully regulated [the quality of the arsphenamine improved and more attention paid to the preparation of the patient]. A purplish swelling of the face, in the course of an over-rapid injection, known as "nitritoid crisis," may be efficiently controlled, as shown by Milian, through injections of $\frac{1}{2}$ mg. of adrenalin, which exerts a a preventive effect. In case the injection be followed in the next few hours or the first days by convulsions or delirium or spasms with a tendency to coma, a profuse venesection should at once be preformed, adrenalin should be injected, a spinal puncture made and intravenous irrigations with physiological salt solution be

administered. The employment of these measures has proved successful in very alarming cases, which, let me repeat, are of altogether exceptional occurrence.

Auxiliary Medication.—Potassium iodide, introduced into the treatment of syphilis by Wallace and by Ricord, for a long time advocated as a specific in tertiary lesions, has lost much of the favor it formerly enjoyed, especially since the introduction of arsenobenzol and since it has been shown to have no influence on the Wassermann reaction. It may be described as not indispensable. In the secondary stage, in daily doses of 1 to 3 grams, it serves to control the headache and the painful manifestations. In the tertiary stage, it is used, combined with mercury in doses of 4 to 12 grams, to stimulate the healing of sluggish lesions and against the lesions of the nervous system or the viscera. Its employment should be avoided, however, in the treatment of so-called parasyphilitic disorders (general paralysis, tabes, etc.) and in the case of laryngeal symptoms. In subcutaneous injections it is too painful, but it may be administered in enemas [and has been given intravenously; but this seems to me a futile procedure]. Other iodide compounds besides potassium iodide are of less efficiency.

Some authors prescribe as a routine procedure a mixed iodomercurial syrup, of the type of Gibert's syrup; it is better to administer the two medicinal agents separately, Sulphur medication, cod-liver oil, rarely iron, more frequently hydrotherapy, cures at watering-places with warm mineral springs, such as Luchon, Uriage, la Bourboule, Amélie-les-Bains [or Hot Springs], or sodium chloride springs, may prove of the greatest value for the improvement of the patient's general condition or his tolerance of mercurialization.

It is always necessary to watch the alimentary, physical, occupational and emotional hygiene of syphilitic patients, cutting out especially the abuse of alcohol, venereal excesses, late hours, and overstrain of all kinds. A moderate use of tobacco may be permitted, except at times where the production of buccal mucous patches is threatened or when it seems to favor the onset of mercurial stomatitis.

General Conduct of the Treatment.—The so-called *abortive treatment* of Metchnikoff consists of rubbing without delay a calomel salve, 1 to 3, into the mucous membrane or an erosion which has been exposed to syphilitic contact. Although it has been experimentally tested in monkeys, this procedure is not free from disadvantages and has not been demonstrated to be superior to simple washing with soap and water. [Prophylactic treatment after venereal exposure is obligatory in the United States army. An experience based on several hundred thousand cases shows that the Metchnikoff inunctions applied within six hours after exposure are a practically certain preventive of infection.]

Early excision of the chancre is certainly not capable, as had been believed, of bringing about an *eradication of syphilis;* when the operation is possible without producing deformities, some authors have found it useful, but there is nothing to show that it modifies the gravity of the infection.

A fundamental rule, which must never be slighted, is as follows: *Never begin specific treatment before the diagnosis has been positively established;* this would be a serious error, which before long would inevitably lead to insuperable difficulties.

When the diagnosis is definitely established, the general specific treatment must at once be instituted, taking care at the same time of the local lesions and guarding against contagion. Recent experience has shown the great importance of a vigorous offensive, striking hard and strong, at least in the primary stage. In all stages of the disease, we must aim not only at extinguishing the actual manifestations, at "bleaching the syphilitic" to use a French expression, but at completely stamping out the virulence of the disease and safeguarding the future—aims which are usually attainable.

The problems dealing with the general conduct of the treatment have been reawakened by the introduction of the arsenical medication. The era of trials and discussions is not yet past, so that no detailed and definite rules can be formulated. I shall therefore restrict myself to describing my actual mode of procedure and that of the majority of experienced syphilographers.

It is quite generally agreed to begin in all cases with a medication which although cautious must be as energetic as the patient can tolerate; and to continue with the tried and approved method of chronic intermittent treatment.

It seems to me highly advantageous to begin the treatment with novarsenobenzol or arsenobenzol. These remedies are positively indicated for patients likely to contaminate their environment, married persons, those expecting to be married, fathers of families, prostitutes, etc.; or in the presence of malignant symptoms or lesions of a destructive character; or when a bad condition of the teeth, which is common, or of the intestinal tract suggests that mercury will be badly tolerated; or in tuberculous patients in whom the breaking down of tubercles is favored by mercury. Inversely, these remedies should be omitted or very cautiously employed in cases of cerebrospinal, meningeal, renal, cardiovascular or hemorrhagic manifestations; under these conditions, it is preferable to rely exclusively upon soluble mercurial injections.

In a general way, in the absence of contra-indications, I consider it advisable to give at first about 5 or 6 injections of novarsenobenzol at a week's interval (not less than four and very exceptionally more than seven) in progressive doses (of 0.30; 0.45; 0.60;

0.75; 0.90); in case of arsenobenzol the doses are one-third smaller and it is better not to go above 0.50. In this increasing dosage, it is of course necessary to take into consideration the signs of possible intolerance, the strength of the patient, the character and more or less obstinate behavior of the symptoms.

Next, after a few weeks of rest, mercurial treatment is begun with soluble injections in a series of 20 to 30 every two or three months; or, as seems preferable to me, with injections of gray oil, in strong doses, in series of 6; three of these series in the course of each of the first two years, another two or three series in the course of the third and again in the fourth year, appear to be a treatment capable of providing adequate security.

The question has been raised why the two powerful antisyphilitic agents in our possession, arsenic and mercury, should not be simultaneously employed from the start. As a matter of fact, this *combined treatment*—which consists in applying during the first series of novarsenobenzol injections, 4 or 5 soluble mercurial injections in the intervening days—is, as a rule, very readily tolerated by robust individuals, in spite of the theoretical fears which have been entertained; stomatitis and albuminuria are of rare occurrence.

It is assuredly rational and advantageous, especially in the presence of some special indication (positive Wassermann reaction) to interpolate in the course of the second to fourth year, one or two series of novarsenobenzol injections between the mercurial treatments.

The same procedure, combined or alternating medication, may be adopted in the treatment of late secondary lesions or tertiary manifestations.

One of the most mooted questions is that of the part played by the *Wassermann reaction* in the conduct of the treatment. Some allow themselves to be guided to a large extent by the modifications of the Wassermann reaction of the bloodserum, periodically tested every three months for instance. Some even go so far as to follow systematically the indications furnished by the reaction of the cerebrospinal fluid. Others go too far in the opposite direction and disclaim all indicative value of these reactions for the treatment. In my opinion, this factor should be kept in mind, but to be guided by the serum-reaction alone would result either in abbreviated treatments when it remains negative, or to unduly prolonged or dangerously increased medications in case it remains positive. It is a matter of judgment and clinical sense.

From a very elaborate review of this question, Hudelo, writing in *Paris Médical*, May 5, 1917, concludes that the following procedure is to be recommended: At the beginning, administer 8 or 9 injections of nov-arsenobenzol at five-day intervals until a total of 4 to 5 grams

has been given, interpolating twenty injections of 0.01 of a soluble mercury salt. After six weeks to two months, if the Wassermann test of the blood is negative, injections of soluble mercury salts are to be given for six months, 10 each month, or 20 every two months; if, on the contrary, the Wassermann reaction again becomes positive, repeat the combined treatment of the first forty days. At the end of the first period of six months, a spinal puncture is made; if it reveals an abnormal condition (hypertension, albuminosis, lymphocytosis, positive Wassermann), the duration of the intervals of rest must be reduced or a prolonged combined treatment be resumed; when clinical symptoms are present (headache, etc.), the doses should be diminished, but the series be prolonged, as recommended by Ravaut. At the end of the sixth month, administer a series of 20 soluble mercury injections, or of 6 injections of gray oil, every three months until the completion of the second year; every four months during the third; every six months in the fourth and in the fifth year. The Wassermann reaction should be verified about once in four months.

In all probability, the new methods of specific treatment will permit an abbreviation of the time-honored delay of four or five years expected from syphilitics who are contemplating *marriage*. It is advisable, however, while taking into consideration the thoroughness of the treatment and the serum-reaction, not to depart too far from the limits established by long experience; as heretofore, the recommendation of a course of treatment with gray oil shortly before marriage is sound advice.

[As the author has well said, the period of trials and discussions has not yet passed. I need make no apology for entertaining very different views in regard to the administration of arsphenamine and I have little hesitation in presenting them even at the risk of some repetition because it is desirable that the reader become acquainted with other views. The final verdict on this subject belongs to the future.

Principles of Treatment.—The fact is indisputable that many cases of syphilis have been cured by a single course of injections of arsphenamine or even by a single injection in the first weeks after infection. In the later stages it requires prolonged treatment with many courses to bring about a permanent Wassermann reaction. This difference probably depends on two factors: the spirochetes have become localized in regions where they are not readily reached by the arsphenamine or they have assumed a form in which they are resistant to the drug, an encysted or spore form.

To meet the indications arising from the possibility of resistant forms we employ the interrupted method of sterilization, the method which long experience has shown to be the best with mercury as the laboratory has shown it to be the best and, depending on the medium, often the only possible method of sterilization. Periods of treatment must alternate with periods of rest during which the organisms may emerge from their resistant stage in order that our sterilizing agents may again successfully attack them in their developed form.

To meet the indications derived from the possibility of remote or inaccessible localization of spirochetes it is necessary to employ the drug in such dosage and to maintain the saturation of the blood with it for such periods that even the tissues least well supplied with a circulating fluid will receive an adequate amount of arsphenamine to kill the germs.

We employ the soluble salts of mercury in daily doses because we aim at maintaining a fairly constant amount of mercury in the system during the period of treatment. We give the insoluble compounds of mercury in weekly doses because with their slower elimination we accomplish the same result with less frequent injections. In rapidity of elimination, arsphenamine is comparable to the soluble salts of mercury. At the end of twenty-four hours only a small fraction of the quantity injected remains in the system. To maintain a saturation of the system for a considerable period it would be necessary to administer arsphenamine in daily doses. This result, however, would not be advantageous even if it were technically feasible because arsphenamine, so far as we know, acts simply as a spirocheticide and a single injection of the drug kills all the spirochetes which it can reach in adequate dosage. There are no spirochetes left in the blood to be acted on by the arsphenamine administered within the next day or two; it is only after the lapse of a longer time that we can again expect to find free spirochetes in the blood. But the repetition of the dose at short intervals for a few days serves to maintain a degree of saturation of the system with the drug which makes it possible for the arsphenamine-laden fluids to reach remote and outlying regions in sufficient dosage.

The principles on which my arsphenamine treatment of syphilis are based are therefore:

1. The principles of interrupted sterilization to destroy the resistant forms.

2. The principle of prolonged saturation to reach and destroy the remote or deeply hidden germs.

Arsphenamine is now made in many chemical factories in different countries. The different products are by no means equally free from impurities which cause immediate or later reactions. Some of these products are followed by dangerous reactions in a large percentage of cases; some are far more free from these dangers even than was the original 606. The general practitioner must choose his arsphenamine in accordance with the experience of those specialists who have larger opportunities for acquaintance with the different brands. The question of cost or of convenience in preparation should have no weight in dealing with so potent a drug and so grave a disease.

Dosage, Preparation of Solution and of Patient. The dose should be proportioned to the weight of the patient, making some allowance for sex and a physical condition possibly weakened by disease, alcoholism, etc. A dose equal to 0.1 for each twenty-five pounds of body weight is the dose for a man; 0.1 to thirty pounds of weight for a woman or a child; and half that amount for an infant. With an otherwise healthy patient the first dose should not be less than the full dose. The employment of a small initial dose followed once a week by slightly larger doses is open to the grave objection that such a procedure is likely to encourage the development of arsenic-resistant spirochetes. The rise in temperature that usually follows the first injection in florid syphilis is of no serious consequence.

For the preparation of the solution it is not necessary to employ a normal salt solution. Freshly distilled and sterilized water in the proportion of 20 c.c. of water for each 0.1 of arsphenamine is a convenient solution. Higher concentrations, 15 c.c. or 10 c.c. of water to each 0.1 of drug may be

used if the needle be of very small caliber so that the rate of flow is lessened; but there is no advantage in this arrangement. The drug must be completely dissolved before the alkali is added and the alkalinization should be made rapidly and exactly. For each 0.6 of arsphenamine a little less than 1 c.c. of a 15 per cent. sodic hydrate should be added in a stream and the remaining few drops necessary for clearing the solution added slowly so that the solution is decidedly alkaline but without needless excess of alkali. The solution is filtered through sterile cotton or gauze into the burette or irrigator which is to be suspended or held about three feet above the point on the patient's arm selected for the injection. The rate of flow is an important factor in the production of nervous and vascular reactions; the needle should not be larger than 20 gauge; the rate of flow should not greatly exceed 0.1 of arsphenamine per minute; the injection of a full dose should require about five minutes.

An examination of the patient's renal function should always precede an injection of arsphenamine. In the presence of a marked nephritis, the dose of arsphenamine must be reduced to one-quarter or one-sixth the normal dose and the effect on the kidneys watched. It is possible that the nephritis is syphilitic and that its symptoms will improve after the arsphenamine. In general, arsphenamine does very little harm to the kidneys, certainly less than a prolonged course of mercury. It is common to find a trace of albumin and a number of renal casts in the urine the day following an injection. I pay no attention to a good trace of albumin or a moderate number of hyaline casts and even a few granular casts. But a considerable amount of albumin and many granular casts, more than eight or ten to the slide, or epithelial casts may be a contra-indication to immediate further treatment. These cases require judgment and careful consideration.

The night before beginning the course of treatment the patient should receive a saline purge. If the treatment is to be administered in the morning at about ten o'clock, he is permitted to take nothing more than a cup of tea or coffee for breakfast before eight o'clock; after the injection he fasts for six or eight hours and may then partake of a moderate dinner avoiding heavy and indigestible food. If the injection is to be administered at two or three in the afternoon the patient may eat his customary breakfast, but should restrict his luncheon to a single cup of tea or clear broth with a small piece of dry toast, not later than twelve o'clock; he fasts for six or eight hours after theinjection and may partakeof a light meal before retiring.

A course of arsphenamine consists of an intravenous injection of a full dose of arsphenamine on each of three successive days. (A course of combined treatment consists of a course of arsphenamine followed by a four to six weeks' course of mercurial injections.) The reactions following this energetic treatment with arsphenamine are not greater than those that occur after a single injection. In fact, it is only the first of the three injections that causes whatever reaction may occur. If the first injection is followed by a severe reaction, nitritoid or toxidermic, it would be folly to give another the next day; but these severe reactions are becoming less and less frequent. It is my growing belief that most of the severe reactions, assuming a reliable arsphenamine, are due to faulty technique: improper preparation of the patient, inadequate alkalinization of the solution, its too great concentration or too rapid administration.

After an experience of nearly five years with this method I can state first, that it is as safe as any other method of employing arsphenamine; and second, that its results are greatly superior to those obtained by other methods of using that remedy. In these statements I have the endorsement of numerous colleagues who have had experience with this method.

Four courses of the combined treatment should be given during the first year after infection, irrespective of the Wassermann reaction. The intervals of rest between the courses last six weeks between the first and second and eight weeks between the others. If the patient came under treatment before the appearance of secondary lesions, the Wassermann reaction will probably have remained negative throughout the year and it may be left to the judgment of the physician whether or not the treatment be continued or the patient simply kept under observation during the next two or three years with Wassermann tests every three to six months. It is of course safer even though it may be unnecessary to continue treatment during the second year with three courses of combined treatment and perhaps two during the third year. In the present state of our knowledge it is impossible to say; there is no absolute criterion of cure except a re-infection. But when the patient has remained Wassermann-negative for a year without treatment and a spinal puncture shows no involvement of the central nervous system, he may be discharged as probably cured. But even then he should be examined serologically at six months' intervals for several years. Consent to marriage, however, may be given.

The syphilitic who comes for treatment in the later stages of his disease should receive three courses of combined treatment each year for three years. Cutaneous manifestations will heal in a few weeks at most under this treatment but visceral lesions do not respond so favorably, especially those of the heart and aorta and the central nervous system. Many of these cases do not become permanently Wassermann-negative. They are nevertheless greatly benefited by treatment and in view of the greatly increased sensitiveness of the present-day Wassermann test it is an open question whether or not much importance should be attached to the persistence of a positive Wassermann reaction in old well treated cases. It is not possible at present to lay down any general rules. Every case of persistent Wassermann-positive should be examined by spinal puncture to determine the possible presence of asymptomatic syphilis of the central nervous system.

Syphilis of the *central nervous system*, especially the early stages of tabes, always demands energetic treatment. In addition to intravenous injections of arsphenamine many syphilographers and some neurologists advocate intraspinal treatment. The treatment devised by Swift and Ellis and modified by Ogilvie is carried out as follows:

The patient receives an intravenous injection of arsphenamine. One hour later 40 c.c. to 50 c.c. of his blood is drawn aseptically from a vein, the blood allowed to clot, its serum separated and placed on ice until next day. It is then centrifuged, one-quarter to one-half milligram of arsphenamine in solution added and the serum inactivated at 56° C. for forty minutes. It is then ready for use. A lumbar puncture is made, an amount of spinal fluid removed equal to the volume of serum, 12 c.c. to 15 c.c., it is intended to inject, then 15 to 20 c.c. of the spinal fluid allowed to flow into a funnel-shaped vessel where it is mixed with the prepared serum and the whole—the spinal fluid plus the serum—is made to flow by gravity into the subarachnoid cavity. The patient remains recumbent a number of hours after the operation. The injections are repeated at two weeks' interval, six to eight injections constituting a course and two courses are given in a year. The value of this treatment is not universally admitted; it is my belief, however, that cases of tabes in which destructive lesions are not far advanced are greatly benefited by intraspinal treatment, symptoms are relieved and the disease checked. In paresis which is clinically recognizable little or no benefit results from the treatment. An early serological or laboratory

diagnosis of paresis has never more than a certain degree of probability. The spinal fluid of the paretic always yields a "paretic gold-sol curve," together with pleocytosis, albuminosis and a positive Wassermann, but similar findings are observed in other, non-paretic, syphilitic affections of the central nervous system and these cases do well under treatment.

In *pregnancy* the problem is to prevent infection of the fetus by keeping the blood of the mother free from active spirochetes. Before the end of the third month of pregnancy when placental circulation is established, energetic treatment with arsphenamine and with moderate doses of mercury lest the kidneys be injured should be administered; after that period arsphenamine and mercury should be given in short courses with short periods of rest; one injection of arsphenamine, four weeks of mercury and one month of rest. In pregnancy in the asymptomatic Wassermann-positive syphilitic of the later stages, the intervals between treatment may be longer.]

YAWS.

Yaws—also known as *pian* and *frambœsia tropica*—is a contagious and inoculable disease of hot countries, which manifests itself by crusted papulo- or pustulo-vegetative eruptions and is caused by a spirochete.

Yaws is endemic and widely distributed in Indo-China, certain parts of the Indies, the Malaysian Islands, Oceanica, equatorial Africa, Central and Southern America.

After an incubation period lasting from fifteen days to six months, the invasion manifests itself by general symptoms, a slight fever, digestive disturbances, headache, bone and joint pains and then by a furfuraceous scaly itching eruption (Fig. 185).

In other cases a primary sore known as the yaws-chancre or the yaws-matrix develops at the site of inoculation, that is, most commonly on the legs or in the face; this lesion may for a long time remain solitary or it may blend with a generalized eruption of the same type. This so-called chancre really has the same objective features as the lesions which follow it (p. 250), frambesia being a monomorphous disease. Glandular enlargement is inconstant. The eruptions spread over the entire integument, lasting several months and following each other during a number of years. The mucous membranes invariably escape. There is no alopecia. Visceral lesions of yaws are unknown.

No human race is exempt from yaws. The disease is inoculable and extremely contagious, even indirectly, but it is not venereal. It is usually contracted during childhood, favored by some excoriation or ulceration which serves as the portal of entry of the virus. Stinging insects may contribute to its dissemination. It is not hereditary. In the course of the first weeks or months, auto-inoculation is still possible; after this time, a relative immunity becomes established, which, however, is not always permanent.

Analogies and a possible relationship between syphilis and yaws

have long been noted, but there are considerable differences: syphilis is universally distributed, is not auto-inoculable, has a prescribed course and polymorphous, non-pruritic manifestations; it frequently involves the mucous membranes and the viscera; moreover, it is hereditary.

The pathogenic agent of yaws, discovered by Castellani in 1905, is the spirocheta pertenuis or pallidula; it is a spiral organism, morphologically very similar to the treponema of syphilis. Noguchi has grown it in cultures. So far it has been found only in the yaws-lesions themselves, but not in the blood and the secretions. Yaws

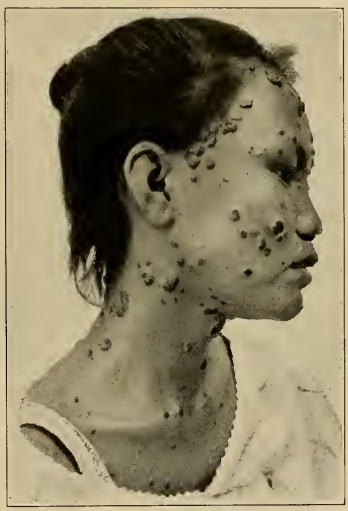


Fig. 185.—Yaws. Courtesy of Dr. E. B. Vedder (Knowles).

is inoculable into monkeys and even into rabbits. It does not confer immunity against syphilis either in man or animals and syphilis on the other hand does not immunize against yaws. But the extract of yaws-papules and the serum of human beings or animals having yaws or cured of the disease, yields a positive Wassermann reaction.

The former modes of *treatment* have been abandoned since the surprisingly rapid and complete action of the arsenobenzols has been recognized. Sometimes one intravenous injection proves sufficient. One month after the introduction of this remedy the large yaws-hospitals in Batavia were closed. In countries where

framboesia tropica prevails, the most trifling wounds must be carefully dressed as they might serve as an entrance-portal for the infection.

LEISHMANIOSES.

Leishmania (R. Ross, 1903) are protozoa related to the trypanosomes, the herpetomonas, etc.; they assume the shape of oval or pyriform corpuscles, from 2 to 4μ in length by $1\frac{1}{2}$ to 3μ in width, containing two chromatic masses known as the karyosome and the centrosome; in cultures they present a flagellate stage. They are found particularly in the endothelia, the leukocytes, the connective tissue and in macrophagic cells and exceptionally in the blood of their hosts.

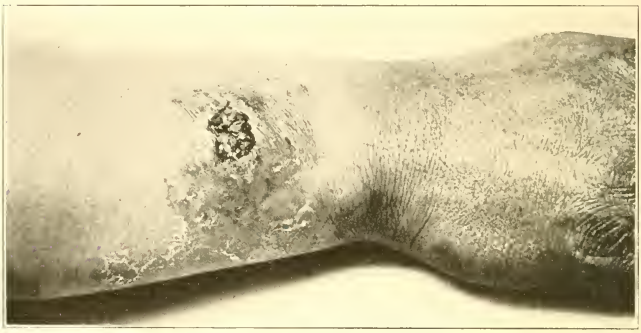


FIG. 186.—Oriental boil, contracted in Biskra.

Oriental Boil (Furunculus Orientalis).—This dermatosis, which is endemic in many subtropical or tropical regions in the north of Africa, Eastern Asia, Central and South America, also bears the names, according to the different countries, of *Biskra boil*, *Aleppo boil*, *Gafsa boil*, *Nile boil* and *Delhi boil; Sulek* or *one-year boil* in Persia, *date-boil*, and so forth.

It is characterized by pustular and ulcero-vegetative lesions which develop without impairment of the general health, most commonly in the fall or during the cool season of the year.

After an incubation of ten days to a month, sometimes longer, a red spot makes its appearance, which becomes papular and pruritic, resembling a mosquito bite; next, the lesion become covered with scales or, as the result of scratching and excoriation, with crusts which conceal a slowly spreading oozing ulcer with a wavy outline (Fig. 186); it frequently has the appearance of a furuncle or crusted

ecthyma before assuming the characteristic features of its adult stage, which have been described elsewhere in this book (p. 250).

The lesions are preferably situated on the exposed parts, the hands, the face and the limbs. As a rule, no more than three or four are present, sometimes only one, very rarely from thirty to forty; they develop in succession and are therefore of different ages. The mucous membranes are seldom affected.

Oriental boil is inoculable and contagious; it confers only a temporary immunity. The blood yields a negative Wassermann reaction.

The pathogenic organism discovered by J. H. Wright (1903) and named by him *helcosoma tropicum* was classified by R. Ross as *leishmania tropica* or *furunculosa*. It is very closely related to, although different from, leishmania Donovani, which causes kala-azar. It is found in thin sections stained with carbolized thionine, within the macrophage cells (Nattan-Larrier); and more readily in smear preparations treated by the methods of Romanovski, Giemsa, Laveran, etc.

It was successfully grown in cultures, in a flagellate form, by Ch. Nicolle, who inoculated it into the macacus. Dogs, camels and horses are said to offer no resistance to it. Without positive proof, it is assumed that the transmission of Oriental boil generally takes place through the intermediation of flies, bugs, mosquitoes or other stinging insects, especially since Nattan-Larrier was able to demonstrate the presence of leishmanias in human blood in the vicinity of the lesions.

The *treatment* consists either in excision of the lesions or in scraping followed by cauterization with the actual cautery or applications of potassium permanganate in powder form. The efficacy of the arsenobenzols is variously estimated. In Brazil, intravenous injections of antimony tartrate are advocated, in doses of 0.05 to 0.10, in sterilized solutions of 1 to 100 or 1 to 500 in physiological salt solution; these injections actually seem to be very efficacious. When the patient leaves the countries where the disease is endemic, cutaneous leishmaniosis tends to heal spontaneously; so that in temperate zones clean dressings may be considered as sufficient. The cure may be hastened by radiotherapy.

Pian-Bois.—This dermatosis is endemic in Guyana among foresters and hunters. Together with de Christmas, I was enabled to observe a case characterized by subcutaneous nodules opening on the skin as ulcers with a granulating floor; the lesions were situated on the back of the hands and arranged at intervals along the lymphatics of arm. The glands become indurated. The course is slow and the disease may recur. Nattan-Larrier (1909) discovered the pathogenic agent which is a *leishmania,* present in rather scanty

numbers, possessing certain special features and named by him the American variety of *Leishmania tropica.*

Boubas or Framboesia Brasiliana.—The disease described under this name by Breda, B. Sommer and others, differs from yaws in its course and also in that it less frequently spares the buccal, pharyngeal, laryngeal, nasal, conjunctival mucous membranes, etc. The initial lesion is a bulla or a pustule, which is followed by a superficial slough; next appears one or several ulcero-proliferative, painless and non-pruritic lesions in patches; the condition may last ten or fifteen years or longer and cause death from cachexia. Children are rarely attacked and the contagiousness is apparently slight.

Boubas were attributed by Breda to a bacillus discovered by him in the lesions; Fiocco succeeded in growing this bacillus in cultures and inoculating it into rabbits. However, Splendore and Carini (1911) have shown that the disease represents a leishmaniosis.

Espundia, described by Escomel, endemic in Peru, Brazil and other parts of South America, begins on the limbs or on the trunk with a nodule which opens as an ulcer with a granulating floor, discharging abundantly and not particularly painful; ulcerative-granulating and destructive bucco-naso-pharyngeal lesions next make their appearance; the duration is twenty to thirty years. Buono de Miranda and Splendore discovered a leishmania which is considered by Laveran and Nattan-Larrier (1912) as belonging to the American variety of Leishmania tropica. [According to Escomel (1919) the lesions of blastomycosis are sometimes associated with those of espundia in the same case.]

Espundia, Bouba, perhaps also Pian-Bois, are therefore probably identical diseases, namely *Leishmaniosis Americana.*

Injections of tartar emetic are recommended as in the treatment of Oriental boil; arsenobenzol is useless.

CHAPTER XXX.

DERMATOSES OF THE LEUKEMIAS AND ANALOGOUS PATHOLOGICAL CONDITIONS.

CUTANEOUS LEUKEMIAS.

In a large number of the skin diseases which have been previously discussed, a change in the cellular composition of the blood can be demonstrated. Not to mention anemia and leukocytosis which are of common occurrence in many infections or toxic diseases of the skin and even in the neurodermatoses, it suffices to point out that a sometimes very considerable eosinophilia is noted in pemphigus and especially in Duhring's disease, where the local eosinophilia is still more marked; in cutaneous hydrargyria, likewise with local eosinophilia (Hoffmann); in leprosy, especially the tubercular form; it has finally been reported in some cases of erythema, urticaria, eczema, etc. However, the relations between these eruptions and the modification of the blood are neither absolutely constant nor probably direct.

The cutaneous affections to be dealt with in this chapter are on the contrary closely related to organic alterations of the blood or, more accurately speaking, with one or other of the hyperplastic diseases of the hematopoietic organs which are grouped under the heading of *leukemias* and analogous pathological conditions. It is therefore indispensable for the dermatologist to be informed on the subject of these general diseases.

Since the investigations of Ehrlich, a distinction is made between two fundamental forms of leukemia: (1) The *lymphatic leukemias* in which the hyperplasia affects the glands and adenoid structures of the same kind, tonsils, closed follicles of the intestine, thymus, etc., including also the spleen; (2) the *myeloid leukemias* in which the lesions affect the bone-marrow and the myeloid tissues, among which the spleen, as an organ of mixed structure, again figures.

These hyperplasias are accompanied by the passage of their constituent cells into the bloodstream, sometimes in enormous proportions, for from 100,000 to over 500,000 white corpuscles per cubic millimeter may be found (*true leukemias*). In other cases, this passage takes place to a limited degree and no more than 20,000 to 30,000 white corpuscles are found in the blood, or even a normal number (*pseudo-leukemias, subleukemias, aleukemias*): but in all

cases the customary proportion of the different kinds of white corpuscles, the leukocyte formula, is modified, so that there may be for example a relative lymphocytosis or submyelemia; all degrees, subject to variations, may be observed in the same case; a subleukemia may even become transformed into a true leukemia.

The leukemias and pseudoleukemias are *chronic* diseases, usually lasting from two to six years or longer; relatively recent observations have shown the existence of a rapid form, leading to death in a few weeks or months and constituting *acute leukemia*.

There is at present a tendency among hematologists to group under the heading of *lymphomatosis* all the hyperplastic affections of the glandular or adenoid system, whether leukemic or not, chronic or acute, generalized or localized; and under the heading of *myelomatosis* those of the myeloid system. The principal forms of these are outlined in the following:

Clinical Forms.—*Chronic lymphatic leukemia* begins insidiously with fatigue, emaciation, hypertrophy of the glands, at first often with a submaxillary, parotid and cervical and later generalized localization, almost invariably associated with an enlarged spleen and sometimes with hypertrophy of the liver. The white corpuscles of the blood, greatly increased in number, are lymphocytes in a proportion of 50 to 95 per 100. Histological examination of the glands and other hematopoietic organs shows an enormous hyperplasia of the adenoid tissue (lymphadenia) which is swollen and packed with practically pure lymphocytes; the other organs and viscera may be the seat either of leukemic infiltration or of true lymphadenomas.

Aleukemic lymphomatosis (also *subleukemic* or *aleukemic lymphadenia*, Trousseau's *adenia*, *Hodgkin's disease*, *pseudoleukemia* of Cohnheim) differs from the above only by the subleukemic or aleukemic condition of the blood. Aside from its progressive and generalized form, there exist localized forms limited to certain groups of glands (neck, mediastinum, abdomen, etc.) or to an adenoid organ (tonsil, intestine), which may develop like a neoplasm and will be referred to later.

Chronic myeloid leukemia, the onset of which is very obscure, manifests itself in its stationary stage by an enormous spleen with a large liver, without glandular enlargement, and by a pale complexion; the blood ordinarily contains about 300,000 white corpuscles the majority of which are granular (polynuclears and myelocytes with neutrophile granules, basophiles and cosinophile ', myeloblasts and nucleated red cells); the bone-marrow is gray or whitish and its constituents are actively proliferating; there is myeloid transformation of the spleen and accessorily a myeloid infiltration of the lymphatic system and the liver; there may be myelomatosis of the

perivascular tissue in the entire organism. The *subleukemic* forms of this disease (submyelemic splenomegalies) are rare and still a subject of controversy.

Acute leukemia, which occurs in a not easily distinguishable lymphatic form and a myeloid form, behaves like an acute infection. The onset is marked by chills, splenic pains, general fatigue and often by angina with persistent fever. The dominating features in the clinical picture consist in the following: Sometimes, moderate glandular hypertrophies, especially at the neck, reaching the tracheobronchial glands with mediastinal symptoms or the mesenteric glands with hypertrophy of the spleen; other cases are associated with hemorrhages, epistaxis, hematemesis, melena, etc., or with buccopharyngeal symptoms, angina, pseudoscorbutic gingivitis; also nervous disturbances, such as headache, vertigo and asthenia. The blood contains over 50,000 white corpuscles, which have reached an imperfect maturity, large lymphocytes or lymphoblasts, or very polymorphous macrolymphocytes. The lesions of the organs are the same as in the chronic forms, but with proliferation and infiltration of large undifferentiated lymphocytes.

Aside from these typical forms of lymphomatosis and myelomatosis, atypical forms are not very infrequently encountered; such as:

Regional glandular lymphadenoma, without leukemia, not easily differentiated from tuberculous adenopathy;

Lymphosarcoma of Kundrat-Paltauf, which presents itself as a malignant glandular tumor, at first regional, with a tendency to become generalized, invading the adjacent organs by infiltration and spreading by metastasis (*lymphosarcomatosis*); it is accompanied by a moderate neutrophilia;

Chloroma (or *green cancer* of Aran), characterized by tumors of the cranial periosteum and lymphomas of a greenish hue in all the hematopoietic organs, with lymphoid or myeloid subleukemia;

Lymphogranulomatosis (of Sternberg-Paltauf), a progressive glandular hypertrophy, capable like lymphosarcoma of invasion and production of metastases, is differentiated from aleukemic lymphomatosis by the presence of yellowish gray points in cross-sections and by a mixed lymphoid and myeloid structure analogous to the structure of wound-granulations, hence its name of granuloma (Benda); it is accompanied by a moderate leukocytosis. This pathological type, which has not been extensively studied in France, corresponds according to Gravitz and others to the majority of cases of Cohnheim's pseudoleukemia and Hodgkin's disease. Several authors interpret it as an attenuated tuberculosis, a genuine glandular "tuberculide."

Etiology and Character.—The etiology of the leukemias and diseases of the same group is entirely unknown.

They were originally supposed to be of neoplastic character and the name of "cancer of the blood" (Bard) was actually suggested for these cases. As a matter of fact, the indefinite cellular proliferation of certain lymphomas and myelomas, the atypical cell-forms met with in certain varieties, the invasion of the vicinity and the metastases, the terminal cachexia, closely simulate the malignant tumors. It is undeniable that the boundaries between lymphadenomas, lymphosarcomas and sarcomas are not very sharp at the present writing.

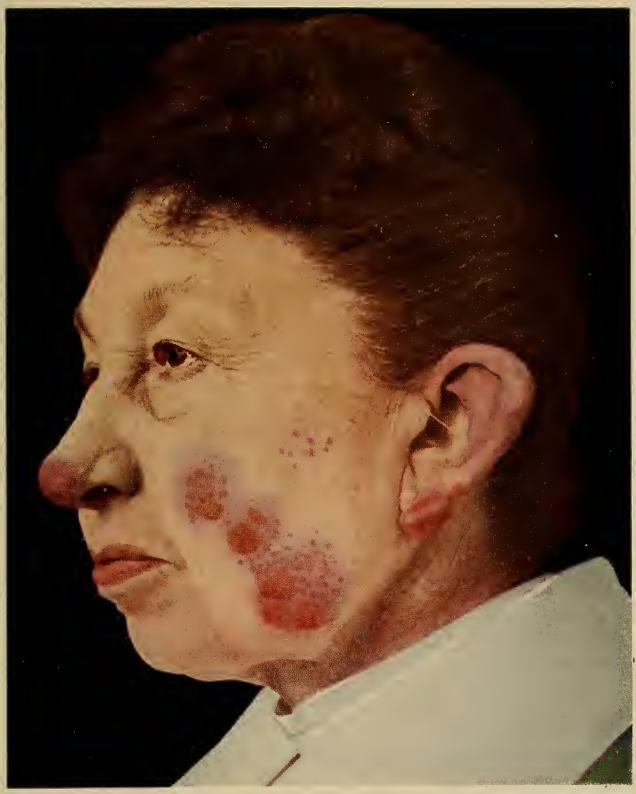
On the other hand, a large array of facts, notably the existence of acute febrile forms of the leukemias is in favor of their infectious character. There is in all probability no reason to look for a "microbe of the leukocythemias" and isolated findings along this line of inquiry have not been confirmed. Cultures and inoculations into animals are generally unsuccessful; in a well-marked case of acute leukemia, I carried out inoculations of blood and tissue-parts into two monkeys without result. But the frequency in the patient's antecedents of various infectious diseases, eruptive fevers, anginas, especially of syphilis and tuberculosis, as well as the analogy in the course of certain lymphomatoses and lymphogranulomatoses with the tuberculous adenopathies are suggestive of a less simple interpretation. Possibly, various attenuated infections or those which develop on an allergic soil, excite a reaction which after being at first defensive, reconstructive and hyperplastic, ultimately becomes atypical, destructive and offensive for tissues of the same kind or those which have been invaded in the vicinity or at a distance; the process, inflammatory at the onset, might in this way acquire the attributes of a malignant neoplasm.

Cutaneous Manifestations.—These may be observed in all the forms, chiefly in the subleukemic or aleukemic lymphomatoses (pseudoleukemias), in chronic lymphatic leukemia, in the acute leukemias, in the case of lymphosarcoma, lymphogranulomatosis, etc.; they are less common in chronic myeloid leukemia, but nevertheless undeniable (Brunsgaard, Lian). They do not differ among themselves according to the type of disease in which they occur; in the very noteworthy and learned contribution of Nanta to the study of the lymphodermas and myelodermas (*Annales de Dermatologie*, 1912), this author agrees with his predecessors in stating that the blood-formula has no bearing on the aspect of the cutaneous lesions.

Besides being extremely variable and multiple, these cutaneous manifestations are often polymorphous in the same case. They may be persistent or transitory, delayed or premature, sometimes preceding the glandular or splenic enlargement and the changes in the blood.

From an objective point of view, they may be divided into three groups:

PLATE III



Leukemic Tumors of the Cheek, Nose and Lobule of the Ear, in a Case of Leukemic Lymphomatosis.

1. Some are of very dissimilar and rather ordinary appearance; representing the *leukemides* of Audry and his pupil Germèr (*Thèse*, 1902). They consist either of persistent pruritus with dryness of the skin; or of pruriginous exanthemas in the form of urticaria, sometimes of papular or vesicular urticaria, simulating polymorphous erythema or Duhring's disease; or the lymphadenic prurigo of Dubreuilh (p. 495) which is accompanied by papules and lichenification. Patches of eczematization also occur, probably due to scratching, as well as pyodermatitides through superadded infection, leaving pigmented macules and small cicatrices. Finally, there may be eruptions of purpura with easily provoked hemorrhages. Histology has only rarely been able to demonstrate miliary lymphomas in certain leukemides, such as prurigo papules.

2. *Leukemic erythrodermas* have been described which do not seem to constitute a generic type. Some cases are perhaps generalized eczemas; others are premycotic erythrodermas (p. 119); finally, some (Nicolau, *Annales de Dermatologie*, 1904) present themselves under the aspect of generalized exfoliative dermatitis or of pityriasis rubra with relative lymphocytosis. In this connection the famous case of Kaposi (1885), entitled *lymphodermia perniciosa*, is invariably quoted; this was characterized by moist and scaly redness, pruritus, a doughy thickening of the skin, followed by cutaneous and subcutaneous nodosities which became ulcerated; hypertrophy of the glands and of the spleen, genuine leukemia, developed rapidly and the patient promptly died; the case was probably one of mycosis fungoides, beginning with erythroderma and terminating as lymphatic leukemia

3. *Leukemic tumors* and *infiltrations* are more characteristic. There exists a localized clinical form, situated anywhere, but often on the face; in these cases, flabby and painless, purplish or brownish-red tumors, with a thin smooth skin, traversed by telangiectases, are seen to develop on the cheeks, the nose and the ears; the swellings slowly increase in size, without a marked tendency to necrosis and ulceration. I have had occasion to follow a case during fifteen years (Plate III). They may be found on the mucous membranes. Sometimes, the condition consists rather of diffuse purplish infiltrations in which vitropressure shows translucid miliary nodules.

Another form presents small red, bluish or dusky tumors, sometimes resembling lepromata. In a case of acute leukemia in a child of thirteen years, I noted very numerous nummular lilac patches, marking the site of not very prominent dermo-hypodermic nodosities distributed over the thorax, abdomen, forehead and scalp.

The structure of these newformations is that of lymphomata or lymphadenomata; small or medium-sized lymphocytes (lymphoblasts or macrolymphocytes) fill the meshes of a fine adenoid network which is supported by the vascular walls.

In other cases, which seem to me relatively less uncommon, there exist multiple cutaneous tumors, some of which are voluminous and occasionally undergo necrosis and ulceration; their structure comprises cells of various types, a considerable number having the characteristics of the constituents of lymphomata or myelomata, although more or less modified and atypical; these cells are enclosed in a network with strands of variable thickness, fairly large in places and provided with connective-tissue cells. These are cases of lymphosarcoma and lymphosarcomatosis, whose relations with the leukemias are doubtful and which closely approximate the sarcomas.

Diagnosis.—In a general way it may be stated that whenever the physician finds himself confronted with a cutaneous manifestation which may be a leukemide, a leukemic erythroderma, or a lymphomatous or myelomatous tumor, he should keep in mind the leukemic and pseudoleukemic states and look for their symptoms.

In the presence of a coexisting progressive glandular hypertrophy or splenic hypertrophy, with corresponding general phenomena and the characteristic lesion of the blood, all doubts are removed.

When no such coexistence is found at the time of the first examination, it is advisable not to discard too hastily the idea of a possible relation between the skin affection and an as yet undeveloped leukemic state, but this should on the contrary be looked for very carefully [by repeated examinations of the blood at intervals of one or two weeks].

This rule is especially imperative in case of tumors of ambiguous character which may be leukemic, lymphosarcomatous or sarcomatous. Laboratory procedures should be called upon to assist the clinical examination; in addition to careful control of the hematopoietic organs by repeated serial hematological examinations, recourse must be had to biopsy, either of carefully selected eruptive lesions or of tumor fragments and possibly of a lymph gland. Fixing agents and stains applicable to the cytological examination of the constituents, notably those of the myeloid series, must be employed.

In attempting to decide between the diagnosis of a lymphoma or a sarcoma, for example, the following considerations must not be lost sight of: the criterion of the "adenoid plexus" is by no means entirely conclusive; it is not always possible to distinguish the cells derived from the blood (hematogenous) from the cells originating locally in the tissues (histogenetic); although it is true in a general way that the constituents of lymphomata are merely infiltrated into preëxisting tissues whereas those of sarcomata replace the latter, this is not a reliable feature since the leukemic constituents are known to possess the power of local multiplication through karyokinesis; one may go so far as to admit, with Dominici and others, the possibility of a primary development of lymphoma in the

skin, as a mesenchymatous tissue, more particularly around its vessels, through a revival of its fetal hematopoietic properties.

It is only at the expense of persevering investigations along this line that we may hope to encounter favorable cases permitting a better understanding of the cutaneous lymphomata and myelomata as well as their relations with the malignant connective-tissue tumors or sarcomata.

MYCOSIS FUNGOIDES.[1]

Mycosis fungoides was first separated and named by Alibert and accurately described by Bazin; it is also known as *cutaneous lymphadenia* of Ranvier, Gillot and Demange and as *granuloma fungoides* of Auspitz. It is a chronic general disease, taking an irregular course, almost invariably fatal, relatively rare and of unknown nature.

Symptoms.—Mycosis fungoides manifests itself by very diverse eruptions and special tumors of an always identical structure.

The *onset*, which is extremely insidious, may take place in four different ways: (1) as a generalized and prolonged pruritus, with nothing to account for it in the beginning; (2) as polymorphous premycotic eruptions, to be described presently; (3) as a premycotic erythroderma (p. 119); (4) as primary tumors.

The polymorphous *premycotic eruptions* are transitory or persistent; they assume the form of erythematous, roseolar, urticarial, circinate or erysipeloid spots or patches; of eczematizations probably due to scratching; more rarely, of purpura, crops of vesicles or bullæ, or of pyodermatitides. Sometimes there are more stationary infiltrated patches known as eczemato-lichenous patches, with irregular outlines, continuous or interwoven, slightly prominent, of a yellowish or purplish red color, which are the seat of severe itching. This surface may be scaly, oozing or crusted; or again, which is more significant, it may be criss-crossed as in lichenization. The number, the distribution and the duration of these premycotic eruptions are too variable to permit of a description.

In the developed stage, the usual findings are as follows: (1) Extensive eczematiform and lichenized surfaces, with edema of the skin, diffusely covering the face which thereby acquires a fairly characteristic leontiastic appearance; nearly the entire integument may be involved, although islands of healthy skin are almost invariably demonstrable; (2) infiltrated patches, of a brick red

[1] In the first edition of this book, this disease is discussed under the infectious dermatoses; this classification, although based upon undeniable analogies, was not supported by proof. I now classify it with the cutaneous leukemias, with which it possesses definite affinities.

color, of variable extent, with an orange-peel or mammillated surface; (3) mycotic tumors (Fig. 187).

The latter, at their onset of the size of a cherry to that of half a mandarin orange, originate on one of the preceding lesions or sometimes in healthy skin; they are more or less soft, of a dark red color, hemispherical, not infrequently constricted at their base, umbilicated and indented, so that they have been compared to a tomato; they may, however, assume a semicircular, crescentic or polycyclic form. They often become ulcerated, through superficial erosion or through central necrosis, while spreading peripherally.

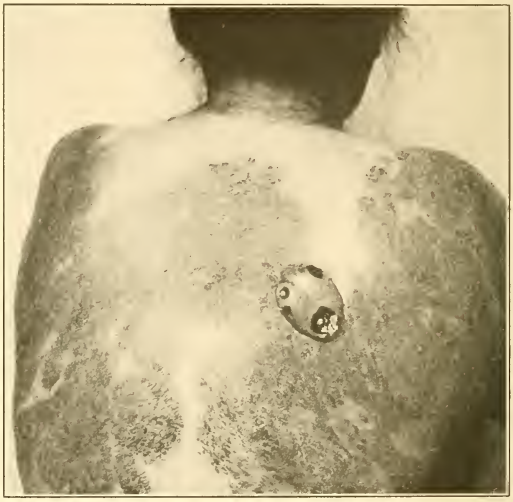


Fig. 187.—Mycosis fungoides. Extensive eczemato-lichenous patches of the back, with partially ulcerated mycotic tumor.

There result enormous ulcerated tumors which may become conglomerated and attain the size of an adult's head, or large ulcers with a ragged, sanious, gangrenous floor, bordered by a fungoid elevation.

It is a remarkable fact that these tumors may at any stage become absorbed and disappear spontaneously, leaving no trace, or being followed merely by a soft white cicatrix with a pigmented areola; other tumors then form in proportion at different points of the body.

They are situated especially on the trunk, the face and on the first segments of the limbs.

The glands are sometimes enlarged early in the disease. From the start any preëxisting nevi become swollen simulating incipient tumors. Alopecia of the affected regions is the rule.

The *course* extends over a period of from two to twenty years, interrupted by spontaneous remissions which may simulate a cure. The patient's strength, complexion, weight and digestive functions finally become impaired and death occurs in marasmus or as the result of a complication.

In the *form with primary tumors* (à tumeurs d'emblée), described by Vidal and Brocq, the tumors are discrete and restricted to one region of the body, originating in healthy skin or on non-pruritic patches, without swelling of the glands; they may become absorbed without ulceration. This type is very closely related to the sarcomata according to Brocq; it also approximates the leukemic tumors.

Pathological Anatomy.—In a contribution of mine to the study of the *premycotic eruptions* (1910), I showed that those which are clinically of ordinary appearance, for example eczematous or lichenoid, have nothing unusual in their histology; the demonstrable changes of acanthosis, papillomatosis, spongiosis, exocytosis and partial parakeratosis are those of eczematization due to scratching. A biopsy at this stage of the disease would therefore not help the diagnosis.

Premycotic erythroderma, on the contrary, has a characteristic structure (Fig. 29); namely, that of the mycotic tumors, but spread out as a thin layer in the papillary body; often the intra-epidermic cellular nests which are peculiar to mycosis are demonstrable, differing through their sharp boundaries from the small islands of spongiosis seen in eczema.

The *mycotic tumors* are uniformly composed of a tissue of lymphomatous appearance, which led the first observers (Ranvier, Gillot, etc.) to the assumption of a "cutaneous lymphadenia." A fine adenoid network, its strands resting upon the vascular walls, is packed with a variety of cells. The great majority of these—rounded or polygonal, approximately the size of a polynuclear cell, with a round or variably deformed nucleus, with more abundant protoplasm than that of a lymphocyte, sometimes containing basophilic granules—seem to be atypical myelocytes of lymphoblastic origin. Furthermore, there are found, in variable proportions, connective-tissue cells, small lymphocytes, large mononuclears, sometimes chorioplasts or giant cells, plasmocytes and mast-cells. Pautrier and Fage in an interesting case encountered a large number of eosinophiles and mast-leukocytes, apparently formed locally at the expense of the lymphocytes. In a general way, there is marked predominance of a certain variety of lymphoid cells, with distinct

polymorphism of the other constituents. Many karyokinetic figures are found.

The epidermis of the tumors is hyperacanthotic at their borders, eroded or missing in ulcerated cases. Sometimes large numbers of "cell nests" (Fig. 29, *a*) are found, filled with lymphoid cells.

The composition of the *blood* is not uniform in all cases and varies in the course of evolution of a given case. A certain degree of anemia and eosinophilia is rather common; neutrophile polynucleosis is not rare; sometimes, a slight myeloid reaction has been noted. The leukemia or leukocytosis which have repeatedly been demonstrated in the terminal stage, is difficult of interpretation.

Nothing very definite is known concerning the character of the visceral lesions which have been found rather frequently in the lungs, the kidneys, the suprarenals and more rarely in the serous membranes, the bone-marrow and the liver of mycotic patients; Brandweiner found metastatic tumors in the brain, with the same structure as those of the skin. This matter requires further investigation.

Etiology and Character.—Mycosis fungoides is known to be neither hereditary nor contagious. It is encountered somewhat more frequently in men than in women, especially between the age of thirty and fifty years. [In the American Dermatological Association's statistics it occurred once in about 3000 cases of all skin diseases.]

The etiology and character of the disease are still problematical. A large array of arguments is in favor of its infectious character, although no parasitological or experimental proof has so far been furnished. To admit its sarcomatous character, with Kaposi, means to strain the analogies and to content oneself with a word. While it is no longer possible to maintain that mycosis fungoides is merely the cutaneous form of lymphadenia, it must be recognized that it is nevertheless most closely related to the lymphodermas, myelodermas and especially the lymphogranulomatoses which are infectious neoplasms. It is especially important to keep in mind cases which are known to have terminated in leukemia. Some day it may be discovered that mycosis fungoides is based upon a reaction of the lymphoid or myeloid apparatus, under the influence of still unknown or variable pathogenetic agents.

Diagnosis.—At the onset a diagnosis may be impossible. The persistence of polymorphous erythematous, eczematous, lichenoid eruptions or of erythroderma, especially when presenting the feature of the edematous infiltration which I have emphasized and when associated with severe itching, should arouse a suspicion of pre-mycotic manifestations. In such cases, biopsy is necessary, but I have already pointed out that the histological lesions are often com-

monplace and possess diagnostic value only in so far as they approach those of premycotic erythroderma.

In the stage of infiltrated patches and tumors, the clinical picture is very characteristic; but before making a diagnosis of such serious import, it should be confirmed by histological examination and blood-analysis. The sero-diagnostic method of complement-fixation with an extract of mycotic tumors serving as the antigen, proposed by Gaucher, Brin and Joltrain, has yielded encouraging but still unreliable results.

Treatment of Mycosis Fungoides and Cutaneous Leukemias.—The periods of spontaneous improvement which are apt to interrupt the clinical course of mycosis and the chronic leukemias make it difficult to estimate the efficacy of the treatment instituted. It is reasonable and entirely indicated to treat the premycotic eruptions, the erythrodermas and the more or less ordinary leukemides locally, according to their eczematous or pruriginous appearance, etc., like the analogous skin affections, often with real benefit to the patient.

Ulcerated tumors should be cleansed, dressed aseptically, tamponed with camphorated naphthol, etc. Sometimes the absorption of tumors and infiltrations has been brought about by means of topical applications of pyrogallol, the employment of which necessitates careful control of the urine; or other reducing agents may be used. Surgical removal cannot be recommended. Mercurial injections have seemed to me to exert a favorable influence in a few cases of premycosis.

The fundamental methods of treatment, however, superior to all others, are represented by arsenic and by radiotherapy. The classical employment of arsenic by the mouth or by intramuscular injections in progressive doses, is at present replaced by intravenous injections of novarsenobenzol; the effect may seem to be favorable, but is more apt to be doubtful or negative.

[In these chronic disorders, the effect of arsenic is developed only when the drug is administered in full doses and over a long period of time. The arsenobenzols exercise their specific effect in syphilis not *qua* arsenic but because of a peculiar molecular structure which renders them spirocheticidal. They are of value therefore only in the spirochetal infections. Their action is necessarily too evanescent to be of use when the metabolic effect of arsenic is the object of its administration. I have found arsenic by daily hypodermatic injections in courses of a month to be of decided value in the leukemias, not only in controlling the early eruptions but also in favorably modifying the blood-picture. I use the formula ℞ sodii arsenatis, phenolis, āā 2.0, aquæ 100.0. Beginning with 0.5 c.c., the daily dose is gradually increased until it reaches 2.0 or 3.0 c.c. at which point it remains stationary for a week, thence declining

rapidly. The course lasts about a month and may be repeated after a two months' interval. The injections are absolutely painless.]

On the other hand, it is unanimously conceded that radiotherapy is the treatment of choice in all the diseases of this group. In different cases the glandular masses, the spleen, the bones, or again the tumors or the various cutaneous lesions are exposed to the rays, with or without filter, in carefully regulated doses, using caution and judgment. The rapidity with which the hyperplasias are reduced and the tumors or infiltrations made to disappear, while the leukocyte formula of the blood becomes modified is very remarkable and has encouraged great expectations. It may be stated that although a noteworthy improvement and sometimes more or less lasting apparent cures are thus obtained, no certain and definite cures have as yet been reported.

CHAPTER XXXI.

TUMORS OF THE SKIN.

Tumors or neoplasms are circumscribed, non-inflammatory new-formations having a tendency to persist and increase in size, of unknown etiology.

The group of tumors was formerly more comprehensive than it is at the present day, for originally all swellings were grouped under this heading; for a very long time, until the discovery of their infectious character, the tuberculomas, syphilomas, lepromas, actinomycosis and so forth, ranked as tumors. In a general way, this is a provisional group; as soon as a neoplasm has surrendered the secret of its origin, it ceases to be considered as a tumor and is aligned with the infectious or other diseases; for this reason I regard as justified the introduction of the words "of unknown etiology" into the terms of the definition of neoplasms.

In a certain number of tumors, although their first cause still escapes us, the pathogenic mechanism to which they are referable can nevertheless be suspected. As a matter of fact, there are altogether only three conceivable mechanisms capable of giving rise to pathological neoplasms. These must necessarily be due to: either an original malformation; or to a reaction of the tissues against external injurious agents, a reaction which is described as inflammatory; or finally to the deposit or retention of elaborated or secreted autochthonous substances.

Among the tumors of the skin, a considerable number belong to the first group, namely the *nevi*.

Others are almost certainly, in spite of the terms of the above definition, of inflammatory or infectious character, as results from their contagiousness or from their structure; such are the warts, molluscum contagiosum, perhaps certain sarcomas, etc.

As retention tumors may be cited the sebaceous cysts, the xanthomas, the tophi of gout; and perhaps urticaria pigmentosa.

As to the majority of neoplasms, however, for example, epithelial cancers, their origin remains as yet inexplicable. Furthermore, it must be kept in mind that in the ultimate analysis the three processes are perhaps neither irreducible nor incompatible. It has actually been shown that imperceptible transitions occur between the inflammatory hyperplasias, the nevus-like malformations and the retention-tumors.

On the other hand from the point of view of their clinical behavior, although some tumors are *benign*, behaving simply like an acquired and persistent local deformity, and others are *malignant*, destructive, invasive and subject to dissemination by metastasis, all intermediate degrees between these two courses are met with, as well as transitions from one form into the other.

On the whole, although it comprises extremely dissimilar affections, this nosographical group of tumors must still be provisionally maintained for the time being.

Several classifications, clinical, anatomical, pathogenic, have been suggested and can be combined to a certain extent. I shall therefore subdivide this class into three orders:

1. *Nevi*, the nevic tumors really belong anatomically to one of the two following groups; but, it seems to me desirable to present a general survey of these malformations, whether they constitute neoplasms or not.
2. *Epithelial tumors.*
3. *Connective- and Vascular-tissue tumors.*

NEVI.

Nevi—popularly designated under the names of *birthmarks, portwine stains, liver spots, beauty spots*, etc.—are congenital malformations of the skin, assuming the form of persistent spots or, tumors. Such is their classical definition.

It has long been known, however, that these deformities are by no means strictly congenital and unchangeable. Many nevi spread, enlarge or diminish in size. Some do not appear until after birth, at the time of puberty or even later. Nor is there any valid reason why the name of nevi should not be applied to certain entirely analogous spots or outgrowths which do not develop until adult life or in old age.

Nevi would therefore be more accurately defined as circumscribed deformities of the skin, of embryonic or developmental origin, appearing at any age and taking a very slow course.

The view according to which nevi are dependent upon emotional or physical disturbances experienced by the mother during pregnancy, rests on no solid basis.

Nevi are of enormous frequency. Individuals entirely free from them are exceptional. The hereditary character of a predisposition for nevi is very obvious and in certain families they are remarkably abundant.

Numerous or large nevi are not infrequently met with in erratic or feeble-minded persons and idiots, so that they have been designated by some as a hallmark of degeneration, an obviously exaggerated statement.

It must be understood that in some cases it is not a question of solitary and as it were accidental products, but of profuse sometimes regional or systematized eruptions of nevi of various types, associated with other malformations: Recklinghausen's disease, lentiginosis, perhaps xeroderma pigmentosum, etc., to which this remark is applicable, may be considered as *nevic diseases*.

Four forms of nevi are recognized: (1) *Pigmentary* nevi; (2) *tuberous* non-vascular nevi; (3) *adenomatous* nevi, in which some of the *cysts* may be included; these will be discussed with the epithelial tumors; (4) *vascular* nevi, which will be found under the heading of angiomas.

1. **Pigmentary Nevi.**—These are brown or blackish spots, without notable thickening of the skin, of variable shape and dimensions; they may apppear at any age but especially about puberty; they are apt to darken or multiply under the influence of pregnancy, uterine disturbances, or exposure to sunlight.

(*a*) *Liver spots*, thus named on account of their color, are round, oval, or polylobular and may exceed the dimensions of the palm of the hand; aside from its pigmentation, the skin is in no way changed.

(*b*) *Ephelides* (p. 323) are not classified with nevi by the majority of authors.

(*c*) *Lentigo* or *lentigines* are brown, black or blue spots, about the size of a lentil, situated on the face, the neck, the shoulders or elsewhere; they are sometimes known in France as "beauty spots." The old writers designated the purely pigmentary flat spots under the name of *nævi spili*. Not uncommonly, however, a lentigo is perceptible to the touch and slightly prominent; all intermediate forms may even be met with between lentigo and soft pigmentary verrucous nevi. In the flat spots of lentigo, the pigment is localized in the epidermis exclusively; when the spots are prominent, pigmentary and nevic cells are found both in the epidermis and in the cutis (Fig. 195).

Exceptionally, the skin is spattered with lentigo-spots in the form of an abundant eruption, constituting *lentiginosis profusa*.

(*d*) *Lentigo maligna* (*infective melanotic freckles* of Hutchinson; *precancerous circumscribed melanosis* of Dubreuilh) is a pigmentary spot which may appear at any age, by no means exclusively in the aged, in any region, although preferably on the face. It has the structure of a diffuse lentigo, spreads slowly at first, then sooner or later gives rise to a nevo-carcinoma and accordingly constitutes an extremely serious affection.

(*e*) In *progressive cutaneous melanosis*, a very rare disease which is more apt to attack the young, a slate-colored or bluish spot is seen to spread slowly, become verrucous, give rise to melanotic infiltration of the glands and lead to death after a number of years as a

result of visceral melanosis. In this form, the pigment infiltrates the connective-tissue cells of the corium.

The *treatment* of liver-spots is like that of ephelides. It would be exaggerated to insist upon the destruction of all lentigo spots, but it is advisable to watch them.

At any rate, it should be kept in mind when treating lentigo, that there is real danger in repeatedly irritating or incompletely cauterizing the spots, for such measures result only too frequently in the production of melanosis and nevus-carcinoma. (French Association for Cancer Research, November, 1913.) Superficial spots can be destroyed with the galvanocautery; carbonic acid snow or electrolysis constitute the treatments of choice. Deeper spots are to be destroyed exclusively by electrolysis. Where there is the slightest indication of extension or malignant change, early wide excision is the only recourse. Radiotherapy and radium are not to be recommended.

[Any method of treatment which does not insure the removal or destruction of every single nevus-cell must be condemned. The cells left in the scars produced by electrolysis or other methods of cauterization are subject to great irritation from the strain of the scar-tissue, and the methods in question necessarily involve the risk of leaving a few cells intact. In practice no harm results in the majority of cases; but I have seen three cases of cancer developing in nevi treated by electrolysis or CO_2-snow. A pigmented nevus should be left in peace or else radically, surgically, removed.]

2. **Tuberous Non-vascular Nevi.**—These are not simply spots but genuine neoplasms of variable dimensions.

(*a*) *Soft verrucose nevi* (*soft warts, cellular nevi*).—This name is applied to elevations of the size of a hemp-seed to that of an almond, more or less prominent, sometimes slightly constricted at their base; the surface is smooth or grained, sometimes hairy; the color is pink, yellowish or dusky. Soft warts may be congenital, but appear more frequently during childhood, increasing in size with the approach of old age. They are especially common on the face, the neck, the thorax and in the neighborhood of the external genitals.

Their chief importance lies in their possible transformation into cancer when they are exposed to repeated irritation or unskilful cauterizations.

Histological examination shows that these nevi consist of an infiltration of the cutis by round or polyhedric cells with a large nucleus, abundant protoplasm, distinctly epithelioid, solitary or arranged in nests, columns or strands, representing the *nevus cells*, which are sometimes pigmented. Virchow interpreted these cells as young connective-tissue cells; Demiéville, as endothelial cells; Unna showed that they are epithelial and derived from the

rete mucosum of the epidermis through proliferation and strangulation of the interpapillary processes. This statement has been verified by numerous authors and I too have been able to confirm it. The epidermis itself is often found to contain nests or clusters of pigmented or non-pigmented nevus cells (Fig. 185). Hence the malignant tumors which arise from these soft warts through proliferation of the nevus cells can no longer be regarded as sarcomas of connective-tissue origin, but rather as epitheliomas of a special kind, *nevo-carcinomata.*

(*b*) *Nevi Molluscum.*—These are more flabby formations than the soft warts, with a thin epidermis and a wrinkled surface. These mollusca may be flattened and spread out, yielding the sensation of a depression in the cutis on palpation; or they may be slightly prominent, like lipomas; more frequently they are pedunculated and are then called *molluscum pendulum.*

The latter, which are very common, develop in the period of adolescence or sometimes in large numbers in the forties; they are chiefly situated on the neck, the back, the eyelids and around the genital regions. Their size varies from that of a pin-head to a pea, often reaching that of a raisin, berry or a small pear. [Crops of these small pendulous fibromata, 50 to 100 or more in number, sometimes develop in women on the face, neck and torso, during the later months of pregnancy, disappearing for the greater part in the course of a year or two, constituting therefore a form of dermatosis of pregnancy.]

Fibroma molluscum; some voluminous nevi molluscum form large flabby tumors, either not very prominent and lobulated or on the contrary hanging down in the shape of a wallet, sometimes reaching the dimensions of an orange or a child's head.

When they enclose hard nodular strands, which usually are thickened nerves, they bear the name of *plexiform neuromas.*

In a considerable number of cases, the large number of nevi molluscum or the mere presence of fibroma molluscum may suggest the existence of an incomplete or abortive form of Recklinghausen's disease which will be described further on.

All these nevi molluscum are histologically made up of a special fibromatous tissue, with fine connective-tissue fibers and very numerous young cells, without an elastic plexus. They practically never undergo a cancerous change [but myxomatous degeneration is frequent].

(*c*) *Hard or Hyperkeratotic Verrucous Nevi.*—These have been described with the circumscribed keratoses (p. 206). Their structure is that of generalized hyperkeratosis; nevus cells are only rarely present.

I repeat that they may assume an arrangement as *linear streaks,*

or as *osteo-follicular keratoses* (p. 403); and finally, the appearance known as porokeratosis, when they affect the palmar or plantar regions (p. 216).

(*d*) *Hairy Nevi* (*Nevi Pilosi*).—An exaggerated development of the hairs, their follicles and their sebaceous glands, may be encountered in the pigmentary nevi and in all the varieties of non-vascular tuberous nevi. These hairs are often thick or enormous, dark and curly, more or less abundant; giant comedos, sebaceous cysts or small horny cysts may also be seen. Hypertrichosis in spots or on larger surfaces, which may constitute a partial furry coat, must be included with the hairy nevi (p. 405).

Treatment.—What has been stated about the treatment of lentigo is strictly true in all respects for the soft warts. Electrolysis is by far the best mode of intervention and yields the best esthetic results. The treatment of the other varieties is optional; according to the cases, they may be dispersed or removed with the galvano-cautery, the bistoury, carbonic acid snow, etc. [I have already stated my objections to any except radical treatment for these conditions.]

Neuro-fibromatosis or Recklinghausen's Disease.—This is a typical developmental and, strictly speaking, nevic disease, sometimes familial, having its principal manifestations in the skin and the hypoderm (Fig. 188).

Neurofibromatosis is characterized by four sets of symptoms: (1) *Pigmentations*, under the form of liver spots, lenticular spots and regional or diffuse melanoderma; (2) *cutaneous tumors*, scattered in one region or over almost the entire body, being nevi molluscum of all varieties and dimensions; they often begin in the hypoderm, where they may be recognized by a bluish spot and by their consistence; they protrude after the fashion of a hernia through the cutis and later on become pedunculated; (3) *tumors of nerves*, assuming the form of hard, rounded or spindle-shaped, sometimes moniliform nodes, perceptible along the course of the subcutaneous nerve-filaments of the forearm, the flanks, the forehead, the neck, or the thighs; they are due to the development around the nerve-sheaths and especially in the interior of the latter, of a tissue like that of fibroma molluscum; (4) *mental disturbances*, consisting of a general intellectual deterioration or loss of emotional control. [In my experience mental disturbances are extremely rare.]

Numerous incomplete cases occur, in which either the tumors of the nerves or the pigmentations, etc., are absent. The pigmentations are of such characteristic appearance, through the association of the three forms mentioned above, that when they are the only symptoms present, a diagnosis of Recklinghausen's disease is sometimes justified.

In other cases, some of the tumors assume a considerable size

and are then designated as major tumors. They present the appearance either of fibroma molluscum or of plexiform neuromata, or even of dermatolyses (p. 372).

Fig. 188.—Neurofibroma. Von Recklinghausen's disease. (Ormsby.)

Neurofibromatosis appears in the course of childhood or adolescence, develops in successive instalments and then persists indefinitely. Distinct retrogressions have, however, occurred in my experience.

EPITHELIAL TUMORS

The tumors derived from the epidermis or its adnexa, hair follicles and glands, consist in part of simple tissue hyperplasias, in part of

hyperplasias with metaplasia or metatypism, *i. e.*, a more or less pronounced modification of the normal cell type. The statement is justified only in a very general way that the former follow a usually benign course, whereas the latter are as a rule of malignant character.

As a matter of fact, the conditions governing the malignancy of tumors, their tendency to indefinite invasion and generalization, are entirely unknown at the present time.

Papillomata.—The attempt has been made to group under this term all outgrowths resulting from a proliferative hypertrophy of the epidermis (p. 237). In reality, the term papilloma is applicable only to an objective appearance. To state that a cutaneous or mucous lesion is a papilloma is no more equivalent to a diagnosis than it would be to describe a disease as a papule or a bulla, but it simply describes an existing dermatological lesion. This dermatological form, to which Chapter XII is devoted, may manifest itself under a great variety of conditions.

Common and flat warts, as well as venereal warts, are almost certainly of infectious origin. The same is true for a considerable number of the other proliferative dermatoses. Verrucous nevi are papillomas resulting from a local malformation.

In the angiokeratomas (p. 695) the verrucous condition is secondary to the angiomatous newformation.

It is an important and noteworthy fact that certain epitheliomas are papillary or begin with a proliferative stage of papillomatous appearance.

Cysts.—Cysts are neoplasms of a special variety and result, not from an abnormal multiplication of living constituents, but from an accumulation of inert secretory products in an epithelial pocket, lined with a connective-tissue membrane; accordingly, they represent *retention tumors*. A distinction is made between two classes:

A. **Epidermic and Sebaceous Cysts.**—Their size varies from that of a millet-seed to that of a hen's egg; they are intradermic or hypodermic. Their consistence may be hard and elastic, or flabby, or even fluctuating. The skin which covers them may be raised or stretched, but usually retains its normal color; it becomes reddened in case of inflammation.

The contents of these cysts are opaque and pasty, made up for the most part of more or less perfectly keratinized epidermic cells, derived from their internal lining, which is epidermic; they also contain fat, a direct product of the developing epidermic cells; fatty acid crystals, soaps, cholesterol and sometimes particles of lime salts. According to its appearance, the contents are described as meliceric, steatomatous, cholesteatomatous, or oily. Suppuration of the cysts caused by infection through pyococci, may lead to a cure.

There are five varieties to be described:

1. *Follicular cysts* and *sebaceous cysts*, generally resulting from a dilatation of the pilo-sebaceous canal. Their first stage is represented by comedo; when more advanced they form little yellowish-white elevations which are superficial, umbilicated, pasty cysts that may be emptied by expression.

2. *Dermoid Cysts.*—These are derived from an epidermic inclusion in the region of certain embryonic clefts and are consequently situated especially at the outer end of the eyebrows, in the neighborhood of the orbit, on the neck, on the perineal raphé, the scrotum, etc. They may contain hair follicles, hairs, sebaceous glands, etc.

3. *Wens.*—These are sebaceo-epidermic cysts, frequently multiple, met with only on the scalp and on the scrotum and appearing only in adult or aged individuals; they are deep and present neither an umbilication nor an orifice. In my opinion, wens are derived from a congenital malformation of folliculo-glandular epidermic buds and should be interpreted as a variety of nevi; constituting *cystic follicular adenomatous nevi.*

4. *Traumatic Epidermic Cysts.*—These are hard, round, painless tumors, which are encountered only on the palmar aspect of the hands and fingers. These cysts seem to result, according to the explanation offered by Gross of Nancy, from a deep implantation of a shred of epidermis, under the influence of a traumatism. They are observed especially in laborers and require months and weeks for their development.

5. *Milium.*—The name of milium, or *grutum*, is applied to white, beady granules the size of a pin-head, which are small epidermic, intra-epidermic or intra-dermic cysts. They are observed as a primary manifestation especially on the upper two-thirds of the face and on the genital organs of both sexes; or secondarily upon cicatrices of no matter what origin; or as a sequel of bullous affections, especially congenital pemphigus with epidermic cysts (p. 191).

Histology shows them to result from the dilatation either of sudoriparous canals or hair follicles. Primary milium is a sort of cystic nevus; the milium of cicatrices is a retention-tumor.

The *treatment* of all these epidermic cysts, if any is required, consists in their removal with the bistoury when they are large or with the curette in the case of milium. Wens may also be treated by injecting into their interior a few drops of pure ether, or ether with bichloride of mercury, or a zinc chloride solution; this injection to be several times repeated. At the time of the spontaneous elimination of the cyst, which takes place at the end of eight or ten days, attention must be given to the thorough extraction of the entire epidermic shell.

B. **Serous Cysts.**—Aside from the hygromas, the branchial cysts of the neck and of cysticercus cellulosa which are not cysts of epidermic origin, this class comprises only a single type:

Hidrocystoma.—Hidrocystoma was pointed out by A. Robinson in 1884 and 1893 as a tense, firm, translucid elevation, from which a watery fluid is discharged on puncture; it is the size of a pin-head or a pea and develops in large numbers on the face, chiefly in middle-aged women who are exposed to the heat of stoves, etc. Hidrocystoma has a tendency to disappear in the winter, reappearing in the spring. The little tumor consists of a dilatation of a sudoriparous canal and is in my opinion of nevic character, therefore constituting *cystic sudoriparous adenomata*. Needless to say, they have nothing in common with dysidrosis and sudamina.

Adenomata.—The name of adenoma is at present applied to benign epithelial newformations, of glandular origin, whose constituents more or less accurately reproduce the texture of the glands from which they are derived.

The adenomas of the skin are subdivided into sebaceous adenoma and sudoriparous adenoma or hidradenoma. All appear to have a congenital malformation for their origin and the name of *adenomatous nevi* is eminently adapted to them.

Adenoma Sebaceum.—The most interesting form is represented by the *symmetrical sebaceous adenomas of the face*. They appear under the aspect of innumerable small tumors, the size of a millet-seed to a large pea, occupying the nasogenial grooves and their neighborhood, the root of the nose and the forehead, the chin, sometimes the vicinity of the auditory meatus, the scalp, etc. (Fig. 189). These adenomas, which are rarely congenital, appear in late childhood, gradually increasing and persisting indefinitely. A white variety, Balzer type, is known, in which the sebaceous glands undergo an atypical proliferation; a red and soft variety, Pringle type, with glandular and vascular hyperplasia; and a hard variety, Hallopeau-Leredde type, in which I was able to demonstrate the predominance of fibrous tissue, so that this condition should rather be described as a fibro-vascular nevus. [Adenoma sebaceum is often associated with other congenital malformations. Several cases of association with multiple subcutaneous fibromas, teratoma of a kidney and mental disorders have been recorded.]

Non-symmetrical sebaceous adenomas are seen in aged persons or adults, scattered in variable number especially on the scalp, the face, or the back, from the size of a lentil to that of a nut or larger.

The *heterotopic sebaceous glands of the mucosæ* may be considered with the adenomata; they are not infrequently seen in the mouth, on the internal aspect of the lips and cheeks. They have the appearance of very small, hardly protuberant spots, the size of a pin-point

or head, of a golden yellow or cream yellow color, solitary or in abundant crops. They do not develop until after puberty. In America this anomaly is described under the name of *Fordyce's disease*. It is of interest only on account of the diagnostic errors to which it may give rise, in the differentiation from buccal lichen planus, etc.

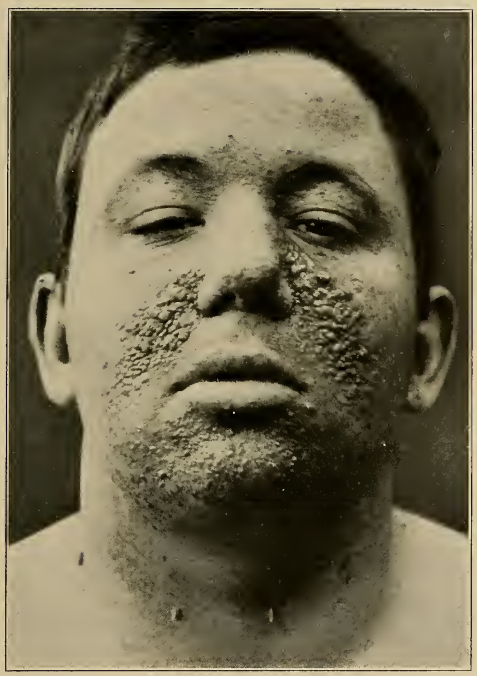


Fig. 189.—Symmetrical sebaceous adenoma of the face, Balzer type.

Hidradenoma.—These small neoplasms—also designated as *syringocystadenoma*, etc.—have two seats of predilection:

1. On the anterior surface of the thorax and the neck, where we described them with Jacquet, under the name of *hidradénomes éruptifs;* they are rare, appearing between the age of ten and twenty years, as numerous solid, often oval protuberances of a pale pink color, resembling syphilitic papules, but not scaly; they

last indefinitely or may disappear. Lesions of the same nature may also be found scattered over the abdomen, on the arms and on the face.

2. *Hidradenomas of the lower eyelids* are certainly much more common. They are met with especially in adult or aged women. They are of the color of the skin, the size of a pin-head (Fig. 190) and must not be confused with xanthelasma of the eyelids.

In both cases the structure of the hidradenomas is characteristic; the corium is found to contain cylindrical and branched epithelial strands, dilated here and there into very minute cysts. They are practically unanimously referred at present to an abnormal proliferation of rudimentary undeveloped sweat-glands. The interpretation implied by the name of *lymphangioma tuberosum multiplex*, given by Kaposi to these small neoplasms, is obviously erroneous.

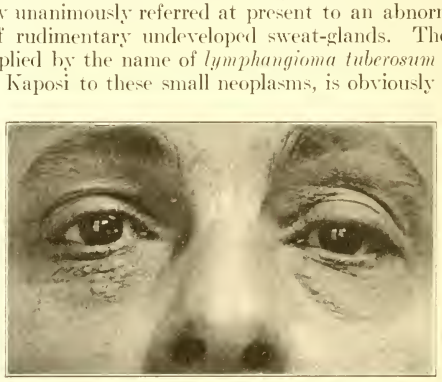


FIG. 190.—Hydradenoma of the eyelids.

The *treatment* of adenomas in general consists in ablation with the bistoury when they are very large. Small adenomas, if the patient desires their removal, should be treated with electrolytic punctures, or when this is not practicable with the curette and the galvano-cautery.

Molluscum Contagiosum.—This designation, due to Bateman—and preferable to the names of *acne variolifórmis*, Bazin; *molluscum sebaceum*, Hebra; *epithelioma contagiosum*, Neisser, etc.—is applied to peculiar small epithelial tumors which are neither adenomas nor epitheliomas.

Molluscum contagiosum presents itself as small hemispherical prominent elevations of a milk-white, pearly or pink color, their essential feature being that they are umbilicated on their crest. Their size varies from that of a pin-point to that of a very large pea; they may become conglomerated into a tumor as large as an almond, which has a tendency to become pedunculated. By compression between two fingers a creamy or pasty mass can be squeezed out

of the umbilicus, consisting under the microscope of horny cells and of refractive ovoid corpuscles, the so-called *molluscum bodies*.

The tumors, which vary greatly in number from a few units to several hundreds, are of variable dimensions and appear insidiously, in successive crops. They are scattered on the face, especially on the eyelids, the neck, the genitals and their neighborhood, but may be found anywhere (Fig. 191). Left untreated they persist indefinitely, without any subjective symptom, multiplying through autocontagion; some of the tumors may become inflamed, suppurate and disappear.

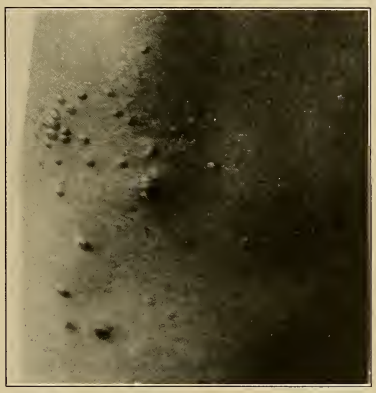


FIG. 191.—Molluscum contagiosum of the region of the knee.

Molluscum is observed [most frequently] in children and in youthful individuals of both sexes with a delicate skin.

Its contagiousness is undeniable; Vidal, Retzius, Hanau and others have successfully inoculated them; the incubation takes several months. [Wile and Kingery have reproduced them by intracutaneous injection of a filtrate (Berkefeld) of macerated molluscum tumor.]

Histology shows molluscum contagiosum to be formed by fairly regular pyriform epidermic lobes with their small extremity turned toward the umbilication. Notwithstanding the gross analogy of their configuration, it is an established fact that these tumors never develop from sebaceous glands. The Malpighian cells of the lobules in proportion as they are pushed back toward the umbilicus, undergo in part a keratinization with eleidine, while others undergo special changes transforming them into molluscum bodies of ovoid shape, which present the reactions of colloid or keratoid substances.

Their special interest lies in the misinterpretation of these bodies as psorospermia or coccidia, a view which has been proved false.

The condition actually is a special dyskeratosis, related to that observed in psorospermosis follicularis and Bowen's disease. The corpuscles are not the carriers of the contagium; Juliusberg and Borrel have shown that the virus of molluscum contagiosum is filtrable.

The best *treatment* is extirpation with the curette, which is easy and leaves no cicatrices. [Expression between two thumb-nails is simple and effective.] Into the small tumors which have first been emptied by expression, the end of a pointed match dipped in tincture of iodine may be introduced. When the tumors are small and very numerous, applications of iodine tincture, soft soap, or camphorated alcohol may prove sufficient.

Epitheliomata.—*Cutaneous epitheliomas* are tumors resulting from an atypical proliferation of the epidermis and of its adnexa. They have also been designated by the names of *epithelial cancers, cancroids*, formerly as *polyadenomas, erosive ulcers, noli me tangere;* abroad, especially in Germany, they are all grouped under the heading of *carcinomata,* whereas in France the latter term is reserved for the malignant and invasive cancers.

In the following brief account I shall include the epitheliomas of the buccal cavity as well as those of the external genital organs, etc., which are equally of dermatological interest.

Among the highly multiple clinical forms of epithelioma, some possess extreme malignancy while others on the contrary are absolutely benign. These will have to be very carefully differentiated. It must be emphasized, however, that even epitheliomas of a malignant character begin as a rule as an apparently insignificant lesion, readily amenable to early treatment. There is no knowledge of greater practical importance for the physician than familiarity with the mode of onset of cancers; when properly forewarned he will know how to caution his patients against the possible gravity of a trifling "sore" and to rid them of it in time.

Clinical Forms.—The attempts which have been made to find in the histological structure of the various forms of epitheliomas an explanation of their widely divergent course have been only partially successful. The classification which I proposed at the International Congress in 1904, although based upon the histological structure, takes largely into account the objective features and the developmental tendencies of each kind, namely those characteristics which possess the greatest practical importance. This classification is here repeated.

1. **Lobular or Spinocellular Epithelioma.**—In this first class, the neoplastic masses are usually arranged in elongated and enlarged

interpapillary buds, or in lobules and in wide strands; they are made up essentially of Malpighian, prickle or spinous cells; these cells undergo the usual epidermic evolution, with formation of keratohyaline, becoming keratinized into horny cells and often into *epidermic pearls*. This class is subdivided into two types:

A. *Superficial Proliferative Type* or *Papillary Epithelioma.*—This type comprises:

(*a*) *Horny papillary epithelioma,* which develops in healthy skin, or not infrequently on a senile keratosis. It is encountered in all regions but especially in the face, on the lips, the neck, the back and on the dorsal aspect of the extremities. It begins as a verrucous elevation and persists a long time in this condition; then, almost invariably in connection with traumatism, it spreads as a protuberant disk bordered by a raised and hem-like margin, the center being studded with villous elevations covered by adherent horny crusts. The glands remain intact for a long time. This epithelioma bleeds readily, may ulcerate and finally leads to cancroid.

(*b*) *Cornu cutaneum*—that is to say, *senile* horny excrescences, for there exists a juvenile form belonging to the class of hyperkeratotic nevi (p. 206)—is a papillary epithelioma with exuberant hyperkeratosis. These horns are of extremely variable size and sometimes resemble a ram's horn from every point of view, including the curvature. They develop in healthy skin or on senile keratosis, chiefly in the face, on the scalp, the glans and the prepuce. Their base may be surrounded by a rose-colored border. Their growth is usually very slow; when they fall out or are removed, they will grow again. The histological lesions are those of papillary epithelioma, more or less pronounced; the horn itself is formed by agglutinated cellular columns; it is softer in its center. These growths may develop into cancroid and this contingency must be kept in mind for the treatment.

(*c*) *Naked papillary epithelioma,* without a horny covering, is met with on the lips, the buccal mucosa, the glans and the vulva; its surface is red, velvety and glistening. It follows a slow and for a long time benign course, but may become ulcerated and pass into cancroid.

It is necessary to guard against confusion of this variety of epithelioma with the proliferative syphilides, with tuberculous lupus of the mucous membranes, etc. Examination by biopsy shows a marked increase of the interpapillary buds in width and in height, with a very moderate cellular infiltration in the upper portion of the cutis.

B. *Deep Type* or *Cancroid Epithelioma.*—This is the penetrating, infective, malignant form of epithelioma of the skin and the mucous membranes with a Malpighian lining.

It develops especially at the orifices; on the lips, on the tongue (Fig. 71) and on the floor of the mouth, where it appears as a complication of leukoplakia, it is known as *smokers' cancer;* it is not uncommon at the anus and on the penis, but may occur anywhere, notably on scar tissue and on lupus vulgaris.

When cancroid does not result from the malignant evolution of a papillary epithelioma, but originates in healthy skin, it is at first a grayish tubercle covered with a scale or a small crust. Under the

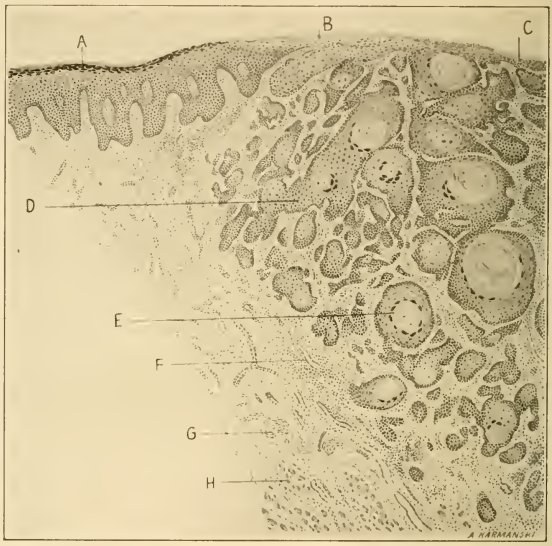


Fig. 192.—Horny lobular epithelioma, spinocellular epithelioma, or cancroid, developing on leukoplakia of the tongue. *A*, leukoplakia; *B*, raised border; *C*, erosion; *D*, lobulated masses; *E*, epidermic pearl; *F*, bloodvessel and plasma-cell infiltration; *G*, nerve; *H*, muscle tissue. × 38.

influence of scratching or traumatisms, it increases in extent and in depth; its crest becomes reddened and ulcerated. Promptly a tumor forms, the size of a cherry-pit or a hazel-nut, hard, imbedded in the skin as well as protuberant. Its borders are raised and swollen and usually more or less hyperkeratotic. On the central area appears first an erosion, then a perpendicular ulceration, which is irregular, fissured, grayish and bleeds easily.

Yellowish granules or filaments, known as "pearls" composed of horny cells and epidermic globules, may be visible and can some-

times be squeezed out. There may be pain on pressure and movement. The glands rapidly become enlarged. I have described elsewhere how this cancer develops on leukoplasic mucous membranes (p. 221).

Histologically, the classical structure of lobulated horny pavement-cell epithelioma is seen (Fig. 192). The large cylinders which penetrate into the depth of the tissues, where they form an irregular lobulated network with large meshes, are apparently derived from interpapillary buds or from pilo-sebaceous follicles. They almost invariably enclose collections of lamellar cells, concentrically arranged after the manner of an onion, known as *epidermic pearls;* more or less numerous dyskeratotic cells (p. 230) are regularly demonstrable. The stroma has a variable structure, but is rather scanty as a rule. At the circumference, plasmocytes are abundantly present. Neoplastic strands having the same structure as the original tumor may be found in the lymph channels.

The tumor ultimately increases in depth, the ulcer becomes gangrenous, the infected cancerous glands may open externally. Death occurs in marasmus or as the result of hemorrhage.

Generalization in the viscera is rare.

The *calcified epithelioma* of Malherbe is a rare and very peculiar variety of spinocellular epithelioma in which the epithelial lobes undergo a total infiltration with lime-salts. It develops as a rule at the expense of old sebaceous cysts, wens or dermoid cysts and consists of masses of stony hardness which grow very slowly. Unless it undergoes transformation into ordinary cancroid, it follows a benign course.

2. **Tubular or Baso-cellular Epitheliomata.**—In this group the neoplastic masses assume a very variable arrangement, as narrow irregularly branching strands, in tubules, in leaf-like lobules with tapering processes, or as a network, etc. (Fig. 193); their continuity with the covering epidermis or with the pilo-sebaceous follicles is often demonstrable. In the center of these masses, small foci of mucous or colloid degeneration are sometimes seen which must not be mistaken for epidermic pearls. The neoplasm is composed exclusively of small oval or spindle-shaped epithelial cells, taking a deep stain, with few or no connecting filaments; briefly, presenting the appearance of the *basal cells* of the epidermis. The stroma is variable, often fibrous and sometimes mucous or embryonic.

This kind of epithelioma is common in all persons of advanced years and in the aged, especially in connection with senile keratosis. Its seat of election is on the upper two-thirds of the face, where four-fifths of all epitheliomas are tubular; it is also encountered, although more rarely, on the lips, the tongue, in the pharynx, on the chest, the genital organs, etc.

The onset is usually in the form of an insignificant papule, which originates in healthy skin or on a patch of senile keratosis; there is a smooth, yellowish or grayish, pearly elevation, firm to the touch, the size of a pin-head or a lentil. It may resemble a flat wart, a cellular nevus, a sebaceous or sudoriparous adenoma. A vague tingling sensation causes scratching; the papule becomes excoriated and covered with a constantly renewed crust or it may remain ulcerated. Its growth is very slow for a few months or even for several years, until quite suddenly it becomes more rapid.

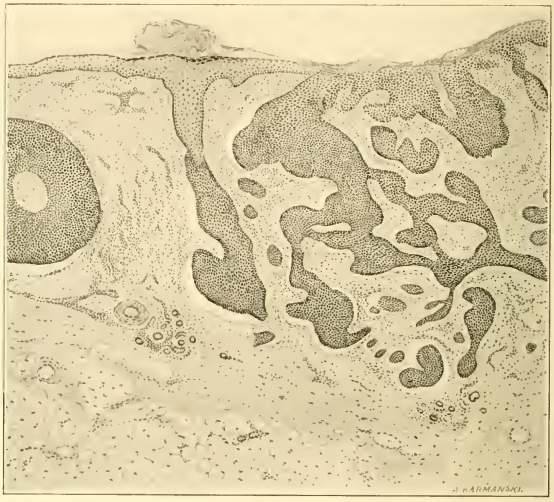


FIG. 193.—Tubular epithelioma (baso-cellular) of the cheek, with the clinical features of flat cicatricial epithelioma. Note the branched epithelial strands, made up of basal cells; their continuity with the surface epidermis and with a bud traversed by a sweat-channel; the erosion covered with a crust on the right side in the illustration; on the left part of a mass which encloses a focus of mucoid degeneration. The neoplasm, which causes no protuberance, occupies the entire cutis down to the level of the sudoriparous glomeruli and the vessels of the subcutaneous plexus. × 38.

Although very readily curable in its incipient stages, by many kinds of treatment, this form of skin cancer nevertheless possesses extreme malignancy; it may become mutilating and incurable when it remains unrecognized, when intervention comes too late or the treatment is inappropriate. Its malignancy is purely local, however, glandular enlargement and metastases being invariably absent.

Although constituting a single histological and nosological species, basocellular epithelioma tends to take an extremely variably course, so that it presents itself clinically under a highly variegated aspect, as tubercles surrounding a cicatrix, as an eroded surface, an ulcer or a tumor; these configurations may moreover coexist or follow one another. The following types may be described.

(*a*) *Flat cicatricial epithelioma* while spreading becomes depressed at its center, which undergoes sclerotic atrophy. After a certain, sometimes very protracted length of time, it presents the appearance of a rounded or rather irregular cicatricial patch, bordered by a seam or chaplet of small grayish, scaly or smooth, more or less translucid elevations, owing to which it is also known as pearly epithelioma. Not infrequently, rather shallow, flat or granulating, slightly bleeding ulcerations form on the borders and gradually invade the vicinity (Fig. 194). The ulcer, bordered or not with pearly granules, often heals on one side while it extends on the other,

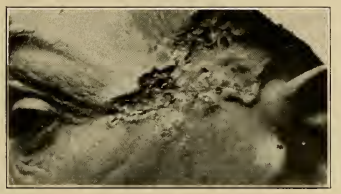


Fig. 194.—Cicatricial flat epithelioma of the temple. Behind, on the side of the scalp and ear, a cicatricial surface is seen, interspersed and bordered with pearly elevations; in front, on the side of the eye, there is a serpiginous ulceration.

destroying the eyelids, the eyeball, the cartilages of the nose and even the bones; it gradually causes enormous and frightful mutilations. I have observed cases which had lasted for twenty and thirty years. The tendency to recurrences, after an apparent cure, is extremely pronounced.

(*b*) *Pagetoid epithelioma* is far from common, but important to know on account of the diagnostic errors to which it is subject; it presents a pinkish surface, distinctly circumscribed by rounded borders marked by a filiform margin, its area being spattered with small scales and crusts, resting upon an atrophic cutis. Its growth is extremely slow. I have observed it only on the face and on the back of aged individuals. It might be confused with senile keratosis, lupus erythematodes, psoriasis and especially with Paget's disease, or with the atrophic spots of Bowen's disease. Biopsy reveals more or less widely separated proliferations of basocellular epithelioma.

(*c*) *Rodent ulcer*—or *ulcus rodens*—is characterized by a shallow and serpiginous ulceration, with a slightly indurated base, without a pearly elevation and a very slow course. Only very fine epitheliomatous tubules are seen in histological sections.

(*d*) *Epithelioma terebrans* may be the outcome of one of the preceding varieties or it may set in primarily; the newformation and ulceration advance in depth rather than superficially; craters and often very deep oozing cavities appear, with a red granular surface, surrounded by a limited induration (Fig. 95). Although very destructive, mutilating and painful, this form likewise possesses a merely local malignancy; the glands generally remain intact as well as the general health. Many months and years may pass before the patient succumbs to hemorrhage or complications of one kind or another.

(*e*) *Proliferative tubular epithelioma*, which is less common, gives rise on the contrary to a genuine tumor, in the form of an eroded and puckered macaroon-shaped elevation, or a protuberance the size of a pea, a hazel-nut, or rarely a large chestnut, sometimes pedunculated, of firm consistence, with an ulcerated and bleeding or crusted surface. This proliferation is sometimes seen to develop rather rapidly at some point of a slowly growing flat epithelioma.

(*f*) *Cylindroma* of Billroth and Malassez, is really an atypical variety of the preceding form in which the stroma undergoes a mucous and hyaline degeneration; there is a formation of translucid cylinders and clear ovoid proliferations which invade and push back the epithelial masses, resulting in peculiar histological appearances which have been variably interpreted, as shown by the numerous denominations of these tumors, such as siphonoma, endothelioma, plexiform sarcoma, angiosarcoma, etc. Cylindromatous tumors are situated especially on the scalp, in the middle of the face, or in the buccal cavity; they are sometimes voluminous often multiple and rarely undergo ulceration. Their prognosis is the same as that of the other tubular epitheliomata.

3. **Nevo-cellular Epitheliomas or Nevo-carcinomata.**—From the purely histological point of view, the cellular nevi themselves are benign nevo-cellular epitheliomas.

These nevi, whether pigmented or not, verrucous or smooth, hairy or glabrous, are sometimes the starting-point of malignant tumors in adult and aged individuals; it has seemed to me that the face and the [heels and] plantar region of the feet are the two elective foci of this transformation.

The tumor then is seen to enlarge in size and becomes painful; its circumference is reddened or becomes the seat of a melanotic pigmentation. Early and more or less rapidly invasive ulceration follows. Similar small tumors promptly begin to multiply in the

vicinity, then at a distance. In case the primary neoplasm was pigmented, generalization takes place by the appearance either of numerous secondary pigmented or non-pigmented tumors or of extensive localized or metastatic pigmentations, or of a generalized *melanosis*. The glands are promptly involved in this form, which often possesses great malignancy; metastases in the viscera, notably in the liver and lungs, is of common occurrence.

The histological structure of these malignant tumors derived from nevi is peculiar (Fig. 195).

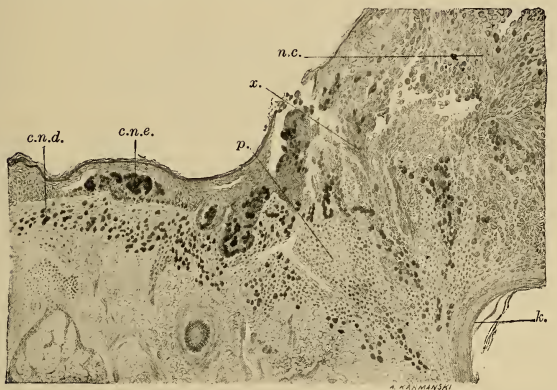


FIG. 195.—Histology of a pigmentary nevus (lentigo) and of the incipient nevo-carcinoma. Section from the border of the tumor. *c. n. d.*, nevus cells, pigmented or clear, in the cutis; *c. n. e.*, pigmented nevus cells in the intra-epidermic tissue; *p*, infiltration of plasma cells at the circumference of the tumor; *x*, transition zone between the nevus and the nevo-carcinoma; *n. c.*, tissue of the nevo-carcinoma of sarcomatous appearance; *k*, portion of an epidermic cyst which was contained in the nevus. × 57.

Their constituents are globular or spindle-shaped, sometimes pigmented, arranged in compact masses, in imperfectly outlined strands or in alveoli; sometimes the appearance is entirely that of a sarcoma.

For a long time such cases were interpreted as sarcomatous tumors and designated as *melanotic sarcoma* or *melano-sarcoma*. The name of *nevo-carcinoma*, proposed by Unna, who showed the epithelial origin of nevus cells and consequently of the neoplasms derived from them, is much better justified (see my paper on nevo-carcinoma, in *Bulletin de l'Assoc. franç. du cancer*, November, 1913).

4. **Secondary Carcinoma.**—*Metastatic* epitheliomas are also met with in the skin, although but rarely, derived from operated or

non-operated cancers of the breast, or from internal cancers, as the result of neoplastic embolisms in the vessels.

They are characterized by hard, pinkish, purplish or brownish elevations, the size of a pin-head to that of a hazel-nut; solitary at first, they gradually become confluent in irregular, mammillated surfaces (Velpeau's *cancer en cuirasse*). They are apt to ulcerate, proliferate and become fungoid. Sometimes an extensive patch or surface of scirrhus cancerous lymphangitis develops, which may resemble a patch of scleroderma.

The histological characteristic of secondary carcinoma of the skin is that the neoplastic masses, made up of cells suggesting the primary tumor cells, are without connection with the surface or folliculo-glandular epidermis; they are arranged in branching tracts following the vascular and lymphatic channels; later on, alveoli are hollowed out. The dermic stroma presents at the onset practically no indication of any reaction, but subsequently becomes sclerotic and retracted.

Etiology and Pathogenesis of the Epitheliomata.—We know that cutaneous epitheliomas practically never develop until after the age of forty years and more frequently in males; when they occur in more youthful patients these will be found to have suffered as a rule from a precancerous affection. The part played by heredity is doubtful.

The cause of the facial localization of the vast majority of epitheliomata of the skin is not known; undoubtedly it is to a considerable extent because this region is the seat of election of senile degeneration and leukoplakia; because it is particularly exposed to traumatisms, to inoculations of infectious germs, to the influence of atmospheric factors and especially of light, which seems to exert a favoring action [and possibly because the complicated planes of embryonic growth in the face afford a ready opportunity for the misplacement of epithelial cells].

The direct cause of epithelioma and cancerous tumors in general still remains unknown.

The parasitic or exogenic theory, although very tempting at first sight, rests on no reliable basis. None of the hitherto described cancer parasites have withstood criticism. The coccidia which were supposed to have been discovered, turned out to be merely forms of cellular degeneration and dyskeratosis. We know that there exists in mice a form of contagious cancer, which is inoculable and can be indefinitely transplanted into animals of the same species, certain breeds of mice being predisposed to it; but the causative agent has never been isolated. There is nothing to justify the statement that human cancers are of infectious origin. Borrel reported in small epitheliomas of the face the usual presence of a demodex, which he believes to be the possible carrier of unknown pathogenic germs (?)

Opposed to this interpretation are the cellular or endogenic theories, which assume aberrant embryonic germs and cellular heterotopia, with Cohnheim and Ribbert, or a loss of balance of the tissue constituents, or their abnormal fertilization, according to Hallion, etc.

It must be admitted that the conception of precancerous states is rather an argument against the parasitic theory. Perhaps, epithelioma is merely the outcome of various processes, sometimes of teratological or dystrophic character; in other cases of ordinary inflammatory or even specific character.

Fig. 196.—Multiple epitheliomatosis of the face on senile keratosis. After a cast in the St. Louis Hospital, Paris.

Precancerous Affections.—This designation is applied to pathological conditions which are so frequently the origin of cancers that this coincidence cannot be the effect of a mere accident.

These affections are of various kinds and have been described in the chapter to which they belong. I shall therefore restrict myself in this paragraph to a general summary.

1. Nevi are malformations which lead to nevo-carcinoma.

2. Various dystrophies are precancerous.

The term of *senile multiple epitheliomatosis* (Fig. 196) serves to designate a very common syndrome consisting of the simultaneous or successive development of several epitheliomas at the site of senile keratoses (p. 209) and on a soil of senile degeneration of the skin. These epitheliomas are, as a rule, of tubular type, sometimes papillary or rarely mixed. Presenile dystrophy is followed by similar sequelæ (p. 358).

Xeroderma pigmentosum (p. 355) and up to a certain point the *chronic radiodermatitides* (p. 454) lead to an analogous picture; in the latter case, the epitheliomas as a rule are horny and papillary, later on cancroid.

Arsenical cancer is simply a progressive and imperceptible transformation of verrucous arsenical keratoses (p. 213) into multiple epitheliomas, usually of a horny papillary type.

3. *Leukoplakia* is entitled to special mention among the precancerous affections, as it is the customary although not constant substratum of the lobulated epitheliomas of the mouth, the genital regions and the anus (p. 221).

4. Epitheliomas of various types likewise develop, although much less commonly, on very different dermatoses, among which must be quoted: lupus vulgaris; cicatrices of any origin, but especially the old cicatrices of burns; the occupational dermatosis of chimney-sweeps, workers with tar and paraffin, coal-heavers; dermoid cysts and wens; ulcers and fistulas, obstinate psoriasis, lupus erythematodes, etc.

5. Among the *dyskeratoses* (p. 230), there are two which evidently represent precancerous affections: In *Paget's disease*, the termination in cancer is the rule, sometimes after a very long time, it must be admitted. In *Bowen's disease* half of the known cases (three of the six cases published by Bowen and by myself) have led to cancerous transformation.

Diagnosis of the Epitheliomas.—The clinical forms are too varied to admit of completeness in this connection. At the onset, the epitheliomas must be distinguished from warts, nevi, etc. Cancroid sometimes resembles syphilitic chancre, or the tuberculo-ulcerative tertiary syphilides, or even tuberculous ulcer. Its neoplastic character must be kept in mind; it is an ulcerated tumor, not an ulceration with an indurated base. Flat cicatricial epithelioma is really easily distinguished from the eczematides, from lupus erythematodes, from tuberculosis verrucosa; more commonly, the question of a tubercular syphilide may arise; but the principal difficulty which occurs in this connection is to decide whether or not a given spot of keratosis is already epitheliomatous.

Under all circumstances, I here repeat the definite and absolute rule which I have previously formulated, to the effect that when there is the least suspicion of epithelioma, recourse should be had to *biopsy* for the certainty which this method alone can supply, thereby permitting the timely institution of appropriate treatment (*cf.*, p. 222).

[The American Society for the Control of Cancer has formulated the rule never to cut into a suspected growth without immediately sealing the cut surfaces by means of the actual cautery; to employ frozen sections for making an immediate diagnosis to be followed by radical operation at the same sitting, if indicated; and under no circumstances to allow more than twenty-four hours to elapse between the biopsy and the operation if the diagnosis is cancer. The risk of dissemination in cutaneous cancers is, however, very small.]

Prognosis and Treatment.—Although the prognosis depends essentially upon the anatomo-clinical type, it is no less true that every epithelioma, no matter what its kind, should be completely removed or entirely destroyed. Internal treatment, arsenic, mercury, etc., is a waste of valuable time; iodide medication is decidedly harmful. Local intervention is what is required. The different forms of epitheliomata, however, are not amenable to the same methods.

Spino-cellular Epithelioma.—Papillary epithelioma must be treated by surgical removal, which is easy and provides rapid, reliable results.

Cancroids demand the earliest possible and very wide surgical excision; as a rule, the corresponding glands must be removed at the same time. Radiotherapy is not applicable to lobulated epitheliomata and is certainly harmful in these cases. The few cured cases of lobulated epithelioma through the *x*-rays or radium which have been reported, leave some doubt in the mind for lack of sufficient histological demonstration of the spino-cellular character of the tumor. On the contrary, I have repeatedly noted the occurrence of frightful aggravations under the action of this treatment. In the case of absolutely inoperable tumors, however, radiotherapy or radium may be utilized for the relief of the pains. Technical improvements of radium therapy and radiotherapy have on several occasions been promised, which would guarantee reliable cures even of spino-cellular epitheliomas; this progress may indeed be hoped for, but for the present a skeptic attitude is justified.

Baso-cellular Epithelioma.—Flat cicatricial epithelioma and *rodent ulcer* are the triumphs of radiotherapy; innumerable cases have thus been permanently cured, with excellent esthetic results. Preference should be accorded to the method of massive doses and filtration through aluminum if necessary, to be repeated, provided the tolerance of the skin permits (p. 453); the treatment should aim in a

general way at keeping the tumor saturated with radiations until after an apparent cure has been obtained. Should relapses occur, they will yield to renewed treatment.

In *proliferative tubular epithelioma*, it is advisable first to excise the larger portion of the tumor, in order to reduce as far as possible the work to be accomplished by the radiations.

X-rays and radium institutes are now so numerous in all countries that recourse to them can almost invariably be had. It is useful to know, however, that baso-cellular epithelioma can be cured by other procedures. Surgical operation is not the best, for in the tubular forms it is necessary to pass rather considerably beyond the borders and the floor of the neoplasm, which leads to deplorable mutilations; in case of a recurrence, the additional operations will involve still greater difficulties. Flat epithelioma can be treated with the curette followed by applications of potassium chlorate, or by thermo-cauterization, or by any one of a large number of caustic agents.

The most convenient and most desirable caustic, which has yielded the largest number of durable and good esthetic results in my experience, is arsenious acid, employed approximately according to the procedure of Czerny and Trunecek. After the epidermised surfaces have been scraped, or freshened or burned with the galvano-cautery, they are painted with a brush dipped in a supersaturated solution of arsenic (arsenious acid, 1 part; water and 90 per cent. alcohol, āā 50 parts); they are then left to dry and are covered with a pledget of cotton. At the end of five to eight days, the crust should be removed; if the subjacent surface is white, it is practically certain that the entire neoplasm has been destroyed; if it is mottled with gray and red, another series of cauterizations should be applied until a perfect result is obtained. This progressive plan of operating provides great security and spares the healthy tissues to the greatest possible degree; the pain is rarely very severe and does not last long.

[Many dermatologists prefer the acid nitrate of mercury to any other chemical caustic. The tumor is vigorously curetted, the bleeding checked by compression for a few minutes and then the full strength Liq. hydrarg. nitratis is thoroughly applied by means of a cotton swab for several minutes. The surface is then covered with a thick layer of powdered sod. bicarb. firmly pressed down. No other dressing need be applied. In ten to fifteen days the crust is shed leaving a clean granulating surface which epidermises with surprising rapidity. The cosmetic result is excellent.]

Other Epitheliomas.—The *cylindromas* are treated with the curette, with the bistoury, or with arsenious acid; recurrences are uncommon.

Nevo-carcinoma has a gloomy prognosis, on account of its tendency to generalization. It is of the utmost importance to intervene early before dissemination has occurred. Electrolysis constitutes the treatment of choice; it sometimes yields unhoped-for results. I am in the habit of inserting the needle its full length underneath the neoplasm, at numerous points from 4 to 5 mm. apart and allowing a current of 3 to 6 milliampères to pass for two or three minutes, using the negative pole. Surgical operation is without advantage.

In *secondary carcinomas*, the prognosis depends much more upon the principal tumor and its glandular and visceral generalization than upon the cutaneous localization. The latter often yield remarkably well to radiotherapy.

Summarizing, it may be stated that in order to treat cutaneous epithelioma to advantage, it is of importance to make an early and accurate diagnosis, not only of the nature, but of the kind of tumor. For this purpose, examination of tissue by biopsy will often be indispensable. The treatment must then be adapted to the type of the neoplasm, to its extent and its depth, as well as to its seat and to the general condition of the patient.

VASCULAR AND CONNECTIVE-TISSUE TUMORS.

This class comprises very different neoplasms, all of which however originate in the last analysis from tissues derived from that portion of the middle embryonic layer known as the mesenchyma. Among these tumors, some have approximately the structure of normal tissues, as in the case of fibroma, lipoma, myoma, angioma, etc. The constitution of others is such as to result apparently from a local oversupply of elaborated material, perhaps not foreign in quality to the normal organism, but undoubtedly so in quantity: to this order belong xanthoma, urticaria pigmentosa and the tophi of gout, which may be considered as retention tumors. Finally, in a third group, a structure is found suggestive of embryonic or inflammatory tissues; in such cases the course may be benign, as in botryomycoma, or, on the contrary, of extreme malignancy, as in the sarcoma.

Fibroma.—Aside from fibroma molluscum which has been mentioned among the nevi, there occur hard dermic or hypodermic fibromata, of very variable size, which may appear at any age; they are sometimes multiple and as a rule do not recur after removal. They are formed by a dense fibrous tissue without elastic network and may undergo fatty, xanthomatous or mucous degeneration, or calcification. Some of these tumors seemed to me to be sarcoids which had become fibrous; others might be regarded as subcutaneous keloids.

Keloids (κηλίς = the claw of a crab) are histologically simply hard fibromas; but they acquire a very special interest through their etiology, their appearance and their clinical course.

Keloids are preferably observed in children and in youthful individuals; their seat of election is on the chest, on the neck and on the ears; they are less common on the limbs. [Negroes are especially prone to keloid.]

A distinction has been erroneously attempted between cicatricial keloids and spontaneous keloids, which represent a single variety of tumors. A keloid may develop upon a cicatrix following a burn (Fig. 197), lupus, chancroid, etc., with the result that the scar assumes a more than hypertrophied and deformed appearance (p. 336); or it may follow a slight traumatism, excoriation, vaccination, perforation of the ear-lobe, the bite of a leech, or the

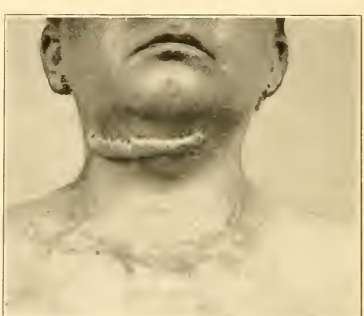


Fig. 197.—Keloid of the neck, on a cicatrix following a burn.

application of a blister, iodine tincture, etc.; it may also appear as a sequel of furuncles, syphilides, or acne pustules. In the last-named case, the post-acneic keloids scattered over the thorax and the face, have nothing in common with the affection known as keloid acne of the nape of the neck (p. 391). The relative rarity of keloids as sequelæ of wounds in the late war is noteworthy.

Whatever its starting-point, a keloid begins as a circumscribed, intradermic prominent induration which enlarges in a few weeks or months. It becomes a very hard tumor, with a smooth, level or indented, pinkish or white surface with steep or gently sloping borders, of globular, oval, or frequently elongated shape; it often exceeds the limits of the original lesion and may attain the size of an egg or form a band or elevation thicker than the finger. Not uncommonly, the borders or the extremities of keloids present

fibrous radiations and sometimes bifurcated strands, which suggested the name given to these tumors by Alibert. There may be absolute freedom from pain, or the patient may complain of unpleasant tingling sensations.

After having increased in size and extent for a few months or years, a keloid may remain stationary or may undergo spontaneous retrogression. Surgical removal is followed by recurrence in case of recent tumors whose development has not been completely arrested. These recurrences are common, not in the entire extent of the surgical cicatrix, but in a portion of the scar, in some of the suture points, but not in all. Moreover, in case of acne for instance, which becomes keloid, some but not all of the pustules undergo this development. From these facts it may be concluded, as I have pointed out, that the appearance of keloids is not connected with a peculiarity of the soil, with a scrofulous or fibroplastic (?) diathesis, as has been claimed, but with a local infection. I am inclined to believe, with T. C. Fox, J. N. Hyde and others, that this infection is of tuberculous character, at any rate in most cases, so that the keloids or at least certain keloids are tuberculides or attentuated tuberculoses. Their structure is that of a very dense fibroma, with mature connective-tissue bundles rich in mast cells (Mantegazza, 1897).

In the *treatment* of keloids, a host of medicinal agents has been utilized—iodine, arsenic, cod-liver oil, salicylates, thiosinamine and fibrolysine; locally, plasters, local douches, scarification, interstitial injections of creosote oil and other substances, electrolysis, etc. I have mentioned the disadvantage of surgical removal, the risk of a larger recurrence with worse deformity. Radiotherapy is more to be recommended. A few sessions will procure considerable improvement; before pushing further, the vitality of the fibrous tissue must be aroused, for example, by negative electrolysis or by local injections, or more practically by deep interrupted scarifications; after which the *x*-rays may be resumed and will be found to have acquired an increased activity. I have obtained numerous permanent results by means of this method.

Lipoma.—The *circumscribed lipomas* belong to the domain of surgery.

Multiple subcutaneous lipomas develop in certain individuals, in crops from a few to several thousands in number. Their structure is that of normal adipose tissue; their size varies from that of a pea to a small mandarin orange; their consistence is soft, lobulated and sometimes pseudo-fluctuating; their distribution is often more or less symmetrical. The differential diagnosis from neurofibromatosis must be made; this condition probably represents an analogous

nevic disease. Fibromas are sometimes partly lipomatous. Certain so-called lipogenic angiomas have a tendency to undergo a transformation into lipoma.

Myxoma.—So-called pure myxoma, soft tumors composed of mucoid connective tissue, are probably partial elephantiasis, as in the case of tumor of the vulva shown in Fig. 116, in which the diagnosis of myxoma had been made. These neoplasms are preferably situated on the genital organs and on the eyelids. Aside from these false myxomas, there also occur myxo-sarcomas or sarcomas in course of myxomatous degeneration.

Myoma.—The dermatomyomas or cutaneous leiomyomas—which alone will be discussed in this place—have been classically described by E. Besnier. These tumors, composed of smooth muscle-fibers forming a network of interwoven bundles, are derived either from the erector muscles of the hair-follicles, or from the muscle cells of the vessels. Myomas are rare and are observed in women more often than in men. They may develop at any point, as disseminated or agminated rose-colored elevations, attaining the dimensions of a pea or at most a hazel-nut. They are frequently very tender on pressure and constitute the majority of what has been described as painful tubercles of the skin; they become the seat of attacks of pain under the influence of local irritation or the action of cold.

Calcareous Tumors.—In addition to true osteomas, which are exceptional, calcified fibromas, calcified epitheliomas, petrified wens and cysts, as well as phleboliths, the following are known:

1. The *petrous tumors* of Poirier, which are calcified fat-lobules the size of a grain of wheat, occurring on the inner aspect of the tibia in aged individuals.

2. *Subcutaneous calcareous granulomas*, which begin as a sort of cold abscess with granular or gravelly contents and may multiply until they lead to death with systemic symptoms. This affection, whose lesions closely suggest those of tuberculosis, is undoubtedly infectious.

3. The subcutaneous calcareous concretions of scleroderma (p. 350).

Colloid Milium.—This very inappropriate term serves to designate small, yellowish, translucid and painless tumors, the size of a pinhead to that of a pea, which may be found scattered or more or less grouped in the face, on the neck and the upper extremities of adults of either sex (p. 359). At first sight they resemble serous cysts; a sort of jelly can be squeezed out of them or they may be enucleated with the curette. Balzer has shown them to be made up of a colloid tissue; degeneration of the dermic tissue into elacine and collacine has been demonstrated. Milian, who recently published a case of

colloid milium (1917), interprets the condition as a limited proliferation of bundles of subepidermic connective-tissue fibers, with hyaline degeneration. The foci stain yellow with van Giesen's stain and a dull blue with thionine. With some attention, it is possible to avoid confusion of colloid milium with hidrocystoma, hidradenoma, lupoma or sarcoid, as well as with senile colloid atrophy (p. 357) which, moreover, is spread out in patches and diffuse. The galvanocautery, carbonic acid snow or the curette may be utilized for the treatment.

Angioma.—Hemangioma.—The majority of angiomas are vascular nevi, the lesion consisting of hypertrophy with ectasis of the veins and, to a less degree, of the capillaries and the arterioles.

According to their clinical aspect and the date of their appearance a distinction is made between several forms:

(*a*) *Flat angiomas* or *flat vascular nevi*, the port-wine stain of popular parlance, are spots of very variable shape, outline and dimensions, punctiform or in very extensive patches. Their color varies at different times from pinkish to bright red or purple. They are especially common on the face and around the natural orifices. On the nape of the neck, at the border of the scalp, they are encountered in nearly 10 per cent. of all persons. [In an examination of several hundred infants less than a week old, I found vascular nevi on the neck below the occipital protuberance in one-third of the cases. Most of these become invisible in the course of time.] They may be seen on the mucous membranes. In epileptics and in feebleminded persons, patches of vascular nevi may be found on the entire body and on the limbs. [I have recorded a unique case occurring in an otherwise normal man in which the entire integument was covered with vascular nevi of the average size of a dime, so closely placed as to form a kind of network. Histologically there was a deficiency of elastic tissue. Dermographism was present.] These angiomas are, as a rule, congenital. They are sometimes hypertrichotic.

(*b*) *Tuberous angiomas* are primary or develop on the basis of the preceding variety. Sometimes they form merely a slightly marked prominence, reducible on pressure, distinctly circumscribed or with diffuse borders; in other cases, they are voluminous and may cause more or less deformity of the lips, the nose, the eyelids, the ears or the tongue, up to lending them a monstrous appearance. The surface is bright red and granular or dark blue and lobulated, according to the depth of the lesions. Those of the cheek and lips sometimes extend to their mucous surface. They often cease very accurately on the middle line. Aside from their disfiguring appearance, these tumors may prove troublesome and after traumatism may bleed profusely.

The *prognosis* and the *treatment* of these angiomas vary. Some of them have a natural tendency to a cure, through lipomatous transformation (lipogenic angioma) or sclerotic change; others simply persist; still others are progressive. In case of a newborn infant suffering from an angioma, its course should accordingly be determined before interfering. In the first two cases, delay is permissible; in the presence of a progressive angioma, prompt measures are required.

For a long time the only choice lay between the following procedures: Compression, usually inefficient; actual cauterization or applications of caustic agents, which leave ugly cicatrices; surgical removal, the method of choice when the dimensions and the seat of the angioma permit; scarifications, of advantage in small flat nevi; vaccination, which is rarely applicable; and finally *electrolysis*. The last-named procedure, the details of which have been well described by Brocq, is preferably carried out with the positive pole and a current of 3 to 10 ma. for one to three minutes; the number of punctures and sessions is increased until a satisfactory result is obtained. [Ultraviolet light with compression often yields good results in the flat varieties.]

At the present day *radiotherapy* is furthermore available, but is not always effective when a safe dosage is employed. *Radium* seems to me to possess a real superiority in this respect, especially in case of tuberous nevi of moderate depth and inconsiderable size; but care must be taken to guard against radium dermatitis. Flat vascular nevi are advantageously treated with carbonic acid snow, which when skilfully handled leaves only slightly visible cicatrices. Large tuberous angiomas belong to the domain of surgery.

(*c*) The *progressive multiple angiomas* constitute a clinical form not heretofore described; I have observed several instances on the face or on the extremities of young or adolescent individuals. They are represented by at first subcutaneous nodosities, of firm consistence, but reducible, which multiply and raise up the skin, giving it a slate-blue hue; later on the skin is invaded and becomes the seat of a purplish depressible growth, which bleeds readily. Ten or fifteen of these tumors, the size of buckshot to that of a large hazelnut, may be seen strung out on the sole of the foot and on the leg, or scattered over the face. The course lasts from six months to two or three years; ultimately, some of these angiomata spread over very extensive surfaces, with diffuse borders, while others remain stationary or undergo spontaneous retrogression. The differential diagnosis may prove difficult from the pigmentary sarcomatosis of Kaposi, from telangiectatic sarcoma, or from nevo-carcinoma, but is established on biopsy, which shows simple cavernous angioma. It is of great importance to treat these angiomata while they are still small with electrolytic punctures, which promptly cure them.

(*d*) *Stellate angioma* is a very common nevus, which often appears late, about the time of puberty or still later. It consists of a red and prominent central point, from which radiate telangiectatic arborizations resembling spiders' feet (Fig. 198). It is easily controlled by the galvano-cautery heated to a dull red, or better by an electrolytic puncture.

FIG. 198.—Stellate angioma or vascular nevus araneus.

(*e*) *Senile angiomas* [in French "pointes rubis," ruby spots] are punctiform or at most lenticular, slightly prominent angiomas which develop with great frequency and in large numbers on the trunk and the limbs of individuals past forty years of age. They have been credited with the property of indicating a visceral cancer, but this has been shown to be erroneous. They may be considered as delayed vascular nevi.

(*f*) The *angiokeratoma of Mibelli* is observed on the extremities of young individuals with acro-asphyxia, suffering from frost-bite, glandular tuberculosis, etc.; so that Leredde classifies it with the tuberculides. The lesions present the appearance of small spots of a bright red color, grouped or agminated, situated especially on the dorsal aspect of the hands and fingers, rarely elsewhere; their surface generally becomes hyperkeratotic and verrucous. After a few months these angiokeratomas may disappear spontaneously. They may be treated like the senile angiomas with the galvano-cautery.

In addition to the Mibelli type, hyperkeratoses have been described as occurring on vascular angiomas and telangiectases of all kinds, notably on the scrotum where they are not very uncommon; I have observed several cases even upon the tongue.

Lymphangioma.—These very rare tumors occur as the result of a newformation of lymph vessels with dilatation. They are referable to a primary malformation and must be considered as lymphatic

vascular nevi. A confusion with lymphatic varicosities has caused much trouble and, as a matter of fact, the distinction between the two groups is not always easy. In my opinion, these differences may be explained as follows:

Lymphatic varicosities, or *lymphangiectases*, are acquired dilatations of the lymph channels of the skin and mucous membranes. They are generally a complication of, or substitute for, elephantiasis (Chapter XVIII) and seem to be caused by an obstruction in a lymph vessel or gland as the result of tuberculosis, syphilis, filariasis and especially of recurrent erysipelas.

Lymphatic varicosities are observed in the mouth, on the mucosa of the lower lip, the cheeks and the tongue, in the form of clear, translucid, beaded or acuminate pseudo-vesicles, of variable number and size; they sometimes become white and opaque; or sometimes red or black through penetration of blood into their cavity. They slightly resemble herpetic vesicles, but are reducible on pressure; moreover, when pricked with a pipette, they furnish an almost unlimited quantity of a clear fluid which is lymph. The subjacent tissues are in a state of chronic edema. Lymphangiectases are frequently associated with elephantiasis of a limb.

Accordingly, lymphatic varicosities may present themselves without tumors, as acquired lesions secondary to a process causing lymph stasis. On the other hand, they will be shown to form also an integral part of the lymphangiomas.

Circumscribed lymphangiomas are congenital tumors or they may appear in early childhood. They consist of rose-colored elevations, more or less firm to the touch, conglomerated in patches and covered with lymphatic varicosities. The latter are very commonly associated with dilated bloodvessels, namely, with hemangioma, and this combination has been interpreted in a variety of ways [and gives to the patches a mottled polychromatic appearance].

Lymphangiomas are preferably situated on the neck and at the root of the limbs; I have observed them also on the flank, in the parotid region, on the knee, etc. They are progressive, but slow and painless, so that the patients put off seeking advice. As a result of interstitial growth, they may give rise to monstrous deformities. The presence of lymphatic varicosities at given points of their surface constitutes their essential distinctive feature.

Lymphangioma may be treated by extirpation, which is rarely followed by recurrence when complete, or with the galvano-cautery, or by electrolysis; the latter is also suitable for the treatment of simple lymphangiectases. Radiotherapy results in a well-marked but transitory improvement.

Primary diffuse lymphangioma is identical with congenital elephantiasis (Chapter XVIII).

Xanthoma.—Under the name of Xanthoma (W. P. Smith) is designated the disease corresponding to Rayer's yellow patches of the eyelids; the vitiligoïdea of Addison and Gull; the xanthelasma of E. Wilson, etc.

Until the last few years, the nature of this disease appeared to be very mysterious; as it could not be considered as neoplastic, it was for a short time believed to be of infectious origin. Its affinities had been recognized on the one hand with diabetes or glycosuria, on the other with chronic icterus and diseases of the liver in general. The yellow cast of the skin and mucous membranes as a whole, which is noted in many cases of xanthoma, not necessarily accompanied by choluria, was distinguished from icterus and described as *xanthochromia;* it has theoretically been referred to xanthomatous lesions of the biliary passages, which, however, have never been demonstrated. Cases of family or congenital xanthoma were known to occur.

Recent contributions, for which we are indebted to Pinkus and Pick, but especially to Professor A. Chauffard with Grigaut and Guy Laroche, have shown xanthoma to be related to cholesterinemia. The fatty substance which is abundantly present in the lesions and characterizes them, is not an ordinary fat, that is, a fatty acid glycerine ether, but is a lipoid, a fatty acid cholesterin ester. The new teachings are accordingly as follows:

Cholesterin, an integral constituent of all body tissues, exists normally in the blood serum in a ratio which, according to Grigaut, oscillates between 1.20 and 1.80 per thousand; it is derived to a small extent from the food but mainly from an internal secretion of various tissues and organs, among which the suprarenal capsules figure predominantly and secondarily the corpora lutea of the ovaries. It is eliminated through the bile, either in its natural state or perhaps in the form of cholalic acid. Its antihemolytic and antitoxic action has been definitely established. Cholesterinemia is increased during pregnancy and in the puerperal state as well as during menstruation; while lowered as a rule at the onset of infections, it is raised in convalescence. Its highest ratio is reached in the course of Bright's disease and especially in icteric conditions due to retention; its relations with diabetes are not constant.

The conclusion has thus been reached that when the cholesterin is insufficiently eliminated it accumulates in the skin (xanthoma) and in the mucous membranes, likewise in the walls of the arteries (atheroma). Xanthematous deposits have sometimes been found on the endocardium, on the peritoneum and even in ovarian cysts (Malassez and de Sinéty). The lesions of xanthoma, being principally due to this deposit of cholesterin, may be considered up to a certain point as retention-tumors.

Clinically, xanthoma presents itself under four forms:

A. *Xanthoma planum of the eyelids*, which has received the special name of *xanthelasma*, is the most common. It consists of straw yellow or dusky, distinctly outlined, sometimes slightly prominent spots which are located more or less symmetrically on the most internal portion of the eyelids (Fig. 199). Their seat, their extent and their color serve to distinguish them from the hidradenomas and the sebaceous adenomas of this region. Xanthelasma usually develops insidiously in adults and aged individuals, somewhat more frequently in women, as a solitary manifestation of xanthoma, often without apparent disturbance of the general health. But I

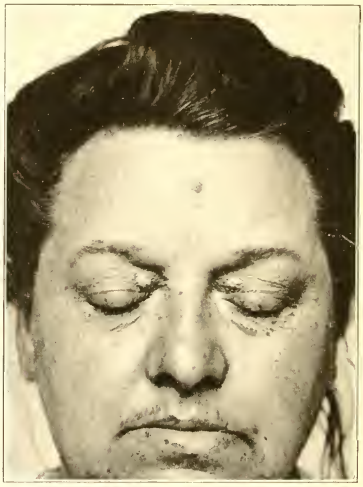
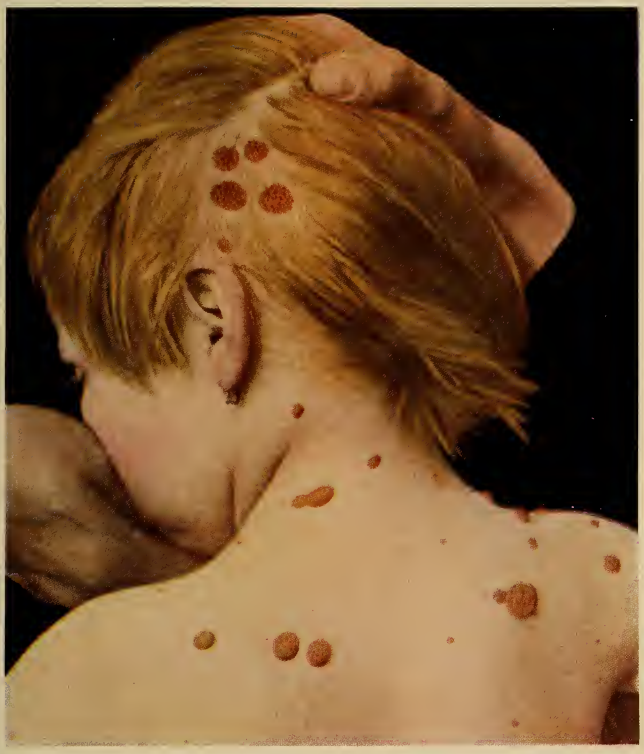


FIG. 199.—Xanthelasma of the eyelids.

have noted its rather abrupt onset in the course of hypertrophic cirrhosis with icterus. It sometimes becomes associated with one of the following forms:

B. *Eruptive xanthoma—xanthoma tuberosum multiplex*—is more uncommon; it may be met with at any age, even in young children (Plate IV). It consists of papular or tuberous elevations, having the dimensions of a pin-head to those of a large bean, of a golden yellow color with a pinkish areola, or it may be of a dark more or less purplish red; in the latter case, the yellow hue of the lesions can be demonstrated by means of vitropressure. Their consistence is sometimes soft, in other cases solid or even keloidal. The eruption

PLATE IV



Eruptive Xanthoma, in a Child of Four Years.

may be slow, progressive, appearing in successive crops; or it may be sudden, becoming established in less than a month; occasionally, the yellow papules make their appearance upon persistent erythemato-urticarial spots.

The lesions vary greatly in numbers in different cases and are sometimes pruritic or painful. They are somewhat symmetrically arranged, especially on the elbows, the knees, the shoulders, the buttocks, the finger-joints and the scalp. Xanthoma may be said to favor high places. This localization is perhaps referable to the fact that these regions are more exposed to blows and to friction, for yellow linear streaks are not infrequently observed at the same time on the flexion-folds of the palmar and plantar regions and the fingers, as well as xanthelasma of the eyelids; in other words, the xanthomatous infiltration preferably takes place at points where the skin is often folded or bruised; Chauffard observed a patient in whom every puncture for arsenic-injection became the center of a xanthomatous nodule.

The lesions persist indefinitely, or they may become absorbed and disappear. There is no reason, however, to distinguish with [M. Morris] Robinson and Török an acute, temporary or intermittent form, constituting *diabetic* or *glycosuric xanthoma*. [The glycosuric form is however clinically distinguished by the presence of a vivid red zone on the sides and surrounding the base of each nodule, an appearance extremely rare in the non-glycosuric form.]

C. *Congenital xanthoma*, in *tumors*, manifests itself in the form of prominent, globular or conglomerated, sessile or pedunculated newformations of a yellow, dusky or purplish color, soft or fibrous and hard, which may attain the size of a mandarin orange or larger. They occupy the crest of the elbows (Fig. 200), the knees, the shoulders, sometimes still other regions. These tumors are present at the time of birth, or make their appearance in the course of the first months of life.

D. *Secondary xanthomatization* has been seen in various tumors, notably in nevi. I was enabled to study a cutaneous fibroma which was histologically xanthomatous, as well as a xanthomatous rhabdomyoma of the tongue; Pollitzer observed one on the eyelids. This cholesterin infiltration of all kinds of neoplasms is very readily accounted for at present.

It seems advisable in this connection to point out that *pseudoxanthoma elasticum* (p. 358), the first reported cases of which were confused with xanthoma, is a cutaneous dystrophy of altogether different character.

Pathological Anatomy.—The blood of xanthomatous patients is far from always being distinctly lipemic, with milky serum. The highest ratio of cholesterinemia noted by Chauffard (1910) in patients having xanthelasma is 1.90 per thousand; in multiple

xanthoma the blood has been found to contain 6,0 or more of cholesterin per thousand.

The *histology* of xanthoma shows that the lesions are made up by collections in the cutis of large connective-tissue cells of very special appearance, polyhedric or spindle-shaped, often arranged concentrically around the vessels, frequently with a compressed central nucleus and a vacuolated foamy protoplasm; their vacuoles enclose fatty granules which will be discussed presently; these are the xanthelasmic cells of Chambard, now known as xanthomatous cells; in certain cases a considerable number of these are polynuclear and giant cells. The xanthomatous cells are arranged in nodules or in strands separated by bands of connective and elastic tissue. There

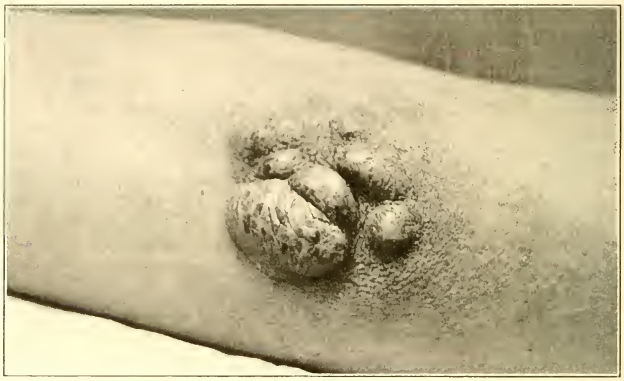


FIG. 200.—Xanthoma in tumors, in a girl, aged nineteen years. Similar tumors existed on the other elbow and on both knees, with xanthelasma of the eyelids.

sometimes seems to be a fibrous reaction which encloses the special constituents and gives to the whole a fibromatous consistence. The epidermis is normal or loaded with pigment. The fatty substance peculiar to xanthoma generally assumes the form of fine granules or round droplets, but sometimes also that of crystals in small rods or in fine needles united in clusters. It is for the most part contained in the xanthomatous cells, although some of it is also almost invariably found in the intercellular spaces. It is soluble in strong alcohol, in ether and essential oils; heat melts it, so that in order to see it, the fresh specimen must be fixed with chromates or better with osmic acid or Flemming's fluid, or with formol; or cut as frozen sections and mounted in glycerine. It will be found that a considerable part of these granules stain poorly with osmic acid, take an orange red stain with Soudan III and, as was first observed

by Stoerk, present the phenomenon of double refraction with polarized light; this is therefore not ordinary glycerine-fat, but rather a cholesterine ether. Other granules are stained a deep black with osmic acid, are turned red by the Soudan reagent and remain dark when the prisms of the polariscope are crossed; these are ordinary fats. The two kinds of granules, which with Policard and Mangini I have usually found to exist together in xanthomas, are variably distributed in the lesions. Histologically the difference between a simple deposit or an active absorption of these substances by the perivascular connective-tissue cells cannot be determined.

[In my opinion, xanthoma tuberosum multiplex, the eruptive disseminated form, and xanthoma planum or xanthelasma, the form that occurs on the eyelids, are two distinct diseases. My studies on this subject have been published in the *New York Med. Jour.*, 1898, *Jour. Cutan. Dis.*, 1910, xxviii, 633, and, in collaboration with U. J. Wile, ibidem, 1912, xxx, 235. I present here the principal points of difference between the two diseases:

Xanthoma.	Xanthelasma.
1. Prominent, hard, round or lobulated tumors.	1. Flat, soft, indistinguishable on palpation, or if at all prominent, feels like a bag of fat.
2. Occurs at any period of life but preferably in early adult life and childhood.	2. Practically unknown before middle age.
3. Development rapid, in a few weeks or months.	3. Development slow, extending over years.
4. Disappears after months or years, or undergoes fibrous changes and persists indefinitely.	4. Persists unchanged through life; never undergoes fibrosis.
5. Extremely rare.	5. Quite common.
6. Distribution general with the neighborhood of the large articulations as seat of predilection. (Under the tense epithelium of the palms the tumors may be spread out in striæ along the normal folds.)	6. Limited to face and neck, the region of voluntary cutaneous muscles.
7. Histologically, xanthoma is an irritative connective-tissue-cell hyperplasia, due to the presence of cholesterol fatty-acid esters derived from the blood. The process begins in the cells of the vascular adventitia, which take up the extruded fatty particles, increase in size and proliferate, sometimes becoming multinucleated. These cell masses in turn commonly act as stimulants to the production of fibroblasts, resulting, in old xanthomas, in the development of fibromas which have erroneously been interpreted as the primary tumor, "xanthofibroma." Xanthoma connotes a systemic disease, a disturbance of metabolism.]	7. No signs of connective tissue or other inflammatory changes. It occurs only when there are striated muscle fibers in the skin, that is, the face and neck (platysma), but similar degenerations have been observed in the tongue, the uvula and in congenital myomata and are reproduced to some extent in the waxy degeneration of muscles after typhoid, etc. The so-called xanthoma cells of xanthelasma are cross-sections or fragments of muscle fibers of the orbicularis palpebrarum which have undergone a peculiar cholesterol fatty degeneration with proliferation of sarcolemma nuclei. Xantho-myoma does not occur; so far from a tumor or increase of the muscle tissue there is, on the contrary, a disappearance of muscle fibers. In long-standing xanthelasma there is scarcely anything left of the original muscle in the areas affected. Xanthelasma is independent of any known general disorder. It belongs with the cutaneous degenerations.]

Treatment.—As a rule, xanthoma shows a tendency to persist and increase. The undesirable spots of xanthelasma and even the xanthomatous papules can be made to disappear, however, almost without cicatrices, by very careful cauterization with the galvanocautery, heated to a dull red. The classical medication with turpentine in capsules, which has long been followed, seems advantageous. At the present day, however, the treatment must obviously be based upon the hypocholesterin diet, recommended by Chauffard, consisting of roasted and broiled meats, green vegetables, skimmed milk, fruits and sugar.

Tophi of Gout.—If the modern interpretation of xanthoma is correct, this disease presents great analogies with gout. The uric acid which circulates in excess in the blood in the last-named dyscrasia, is deposited not only in the joints and the peri-articular tissues and sometimes in the viscera, but frequently also in the skin. The urates deposited in the cutis and the cellular tissue are known as *tophi*. According to Garrod and Charcot, who made a special study of the subject, tophi are found in nearly one-half of all gouty patients (16 times in 37 cases).

Their seat of election is on the external ears, in the groove or on the sharp border of the helix, where these concretions, from one or two to about ten in number, constitute small tumors the size of a millet-seed to a pea. Their color is normal or purplish, with an opaque white hue shining through it. Tophi have occasionally been observed in various other regions; on the alæ or on the bridge of the nose, on the scalp, on the eyelids, etc.

Their other common seat is in the vicinity of gouty joints, over the olecranon or the prepatellar bursa for example, or around the joints of the fingers and toes. These tophic concretions are subcutaneous or intradermic; at first small and multiple, they tend to become agglomerated and spread out. Soft at first, they subsequently become extremely hard.

Tophi make their appearance as a rule after attacks of gout, causing slight inconvenience and sometimes disappearing spontaneously; in other cases, abscesses develop, or still more frequently the skin becomes stretched, opens without suppuration and permits the escape of a chalky substance chiefly composed of sodium urate. Under the microscope it appears as circular crystals, soluble in hot water, which on treatment with acetic acid yield uric acid crystals; with nitric acid and ammonia, the murexide reaction is obtained.

In contradistinction to lime concretions, urate concrements are transparent to the *x*-rays. It has been suggested to treat them by means of lithium ionization. The simplest way to get rid of tophi on the ears is extirpation with the curette, under local anesthesia.

Urticaria Pigmentosa.—The name given to this affection by Nettleship is not a good one, in so far as it leads to confusion with pigmented urticaria (Chapter II).

Urticaria pigmentosa (xanthelasmoidea of T. C. Fox) is not a variety of urticaria, but a chronic skin disease which histologically is ranged with the retention tumors.

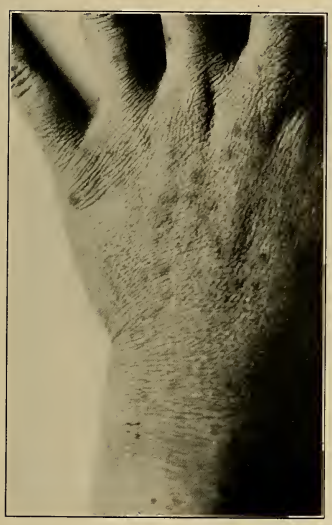


Fig. 201.—Urticaria pigmentosa in the resting state.

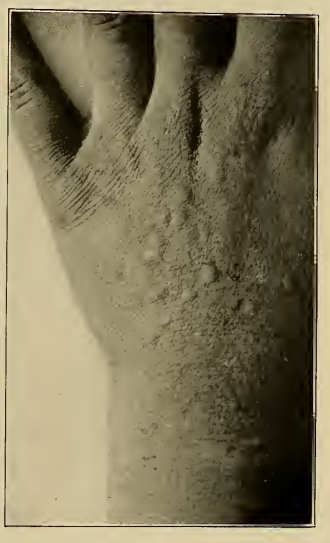


Fig. 202.—The same urticaria pigmentosa in the urticarial state, after irritation by energetic rubbing.

It is characterized by spots or not very prominent elevations, from a pin-head to a fingernail in size, of a dusky or tawny color; these are distributed over the integument in variable number, from a dozen or so to several hundreds, situated especially on the trunk and on the limbs, but sometimes also on the head and the extremities.

The pathognomonic sign of urticaria pigmentosa consists in the inherent property of these spots or elevations to become congested, swollen, firm and distinctly urticarial under the influence of active scratching or pressure with a blunt instrument (Figs. 201 and 202).

This sign by itself alone would suffice to differentiate these lesions from those of lichen planus, psoriasis, syphilides or tuberculides, or from simple macules which they sometimes closely resemble.

The usual teaching is to the effect that urticaria pigmentosa begins a short time after birth, rarely after the first year; that it disappears at the end of eight or ten years through progressive obliteration and would accordingly be very rare in adults. This is often correct. I have shown, however, before the French Dermatological Society in 1905, that this affection may last indefinitely; that its appearance may be noted at puberty or even at a mature age; that it is sometimes familial. The eruption in some cases is subject to congestive attacks with pruritus, spontaneous or in connection with sweating; in other cases, it remains sluggish and latent, so that its existence may be overlooked on superficial examination. The existence of dermographism has been reported in these patients; this may be a mere coincidence and at all events is not common.

The *histology* of the lesions shows in the cutis an abundant infiltration of mast-cells, staining a purplish red with polychrome blue; these cells are spindle-shaped, oval or polygonal when accumulated in masses. Pigment is found in the basal layer of the epidermis and in the papillary body.

The etiology is unknown. Cutaneous irritation, nervousness and emotional disturbances have been held responsible. It seems to me that auto-intoxications of digestive origin or certain disturbances of the hepatic function may play a role. Investigations aiming at the separation of the complex of hepatic insufficiency and biliary retention may help to elucidate this problem, as in the case of xanthoma. If I am mistaken in this hypothesis, urticaria pigmentosa will have to be considered as related to the nevi.

All treatment hitherto attempted, including hot and cold hydrotherapy, radiotherapy, phototherapy, electrolysis, etc., has on the whole proved useless. All that can be done is to prescribe a correct hygiene.

Botryomycoma [*Granuloma pyogenicum*].—This designation is applied to certain benign tumors, or rather persistent inflammatory products which assume the behavior of tumors, possessing very peculiar morphological and histological features.

There is sometimes seen on the hands or on the fingers, or on the sole of the feet, a small soft prominent elevation, bright red in color, smooth or resembling a raspberry, the size of a pea to that of a large hazel-nut; its essential feature consists in its being strangulated at the base, or even plainly pedunculated, as may be verified in doubtful cases with the help of a stylet or a hook. Less commonly, similar growths have been observed on the leg, the forehead, the lips and elsewhere. As a rule the patient will remember some puncture or wound which he has sustained at this point and which has been suppurating for a few weeks or months previously. He will

generally admit having repeatedly cauterized or irritated it in various ways. These tumors may persist for years (Fig. 203).

On histological examination they are found to consist of inflammatory or embryonic connective tissue with large and abundant newly formed capillaries; briefly, showing the structure of *chronic "proud flesh"* [granulation tissue]. The epidermis is missing on their surface and usually stops at the level of the pedicle or slightly above it.

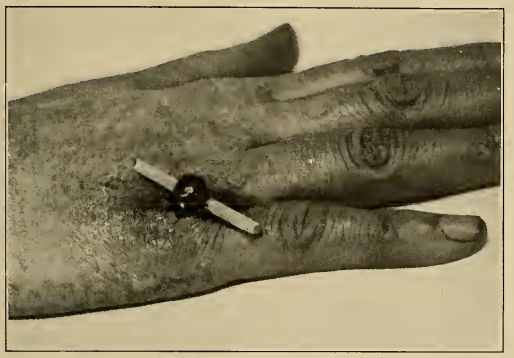


FIG. 203.—Botryomycoma of seven months' standing irritated by traumatism and applications of nitric acid.

The interest aroused by these peculiar small products and the inappropriate name they bear are due to the statement of Poncet and Dor, in 1897, to the effect that these inflammatory growths contained mulberry-shaped hyaline globules, sometimes visible to the naked eye, similar to those which had been described by Bollinger under the name of *botryomyces* in the fungoid growths following castration in horses; it is now known that this appearance is not due to a parasite, but to cellular degenerations of pycnotic type.

Moreover, the coccus growing in yellow cultures which can be isolated from human as well as animal botryomycoses possesses no special features and is not entitled to the name of *botryococcus ascoformans*, applied to it by Kitt; it is nothing more nor less than the Staphylococcus aureus. It would therefore be justifiable to group the tumors caused by it under the heading of pyococcal dermatoses.

[The name Botryomycoma, based on an erroneous pathogenesis, is obviously objectionable. The term granuloma pyogenicum, proposed by Hartzell, is to be preferred.]

The treatment of these newformations consists in their complete ablation [followed by cauterization]; there is no danger of recurrence. [Nevertheless, unless they are thoroughly cauterized they often recur.]

SARCOMA.

Sarcomas are connective-tissue tumors of embryonic structure, usually possessing great malignancy.

On the skin are observed: *primary idiopathic sarcoma,* which alone will be considered here; and *secondary metastatic sarcoma,* derived from the generalization of sarcomatous tumors of the viscera, the glands or the bones.

The primary cutaneous sarcomata belong to different varieties. From the histological point of view, a distinction can be made between the following:

1. *Round-cell sarcoma,* which consists either of small round cells or of large round cells.

2. *Spindle-cell sarcoma,* with spindle-shaped or fasciculated cells.

3. *Atypical sarcoma with polymorphous cells* which were erroneously described by me in former publications and in the first edition of this book, under the name of *lymphosarcoma.*

The so-called *pigmented sarcoma* is a nevocarcinoma (p. 682) which surgeons persist in calling *melanotic sarcoma.* The *multiple idiopathic pigmented sarcoma* of Kaposi (1872) is a separate affection, probably of infectious origin.

A. **Typical Sarcoma.**—These tumors are characterized by their being composed almost exclusively of embryonic connective-tissue cells, all of the same type, round or spindle-shaped in different cases, not contained in an adenoid network and whose bloodvessels are lacunar, in the sense that their walls are formed by the tumor constituents themselves.

This neoplasm of relatively homogeneous structure invades the neighboring tissues by substitution, not by interstitial infiltration; metastases occur by way of the bloodvessels rather than by the lymphatic route. These features are not invariable, however, for any sarcoma may present some rare dissimilar constituents, chorioplaxes, giant cells, etc., as well as points of interstitial infiltration on some of its borders.

Spindle-cell or *fascicular sarcoma,* with large or small fusiform cells arranged in interlacing bundles, presents itself clinically under the aspect of a hard, indistinctly outlined dermo-hypodermic tumor, of a dark red or purplish color, interspersed with telangiectases; it grows slowly and finally becomes superficially ulcerated; it has only a slight tendency toward glandular or visceral metastases, but usually recurs promptly after surgical removal.

Round-cell sarcoma, with small or large round cells, develops more rapidly and usually becomes generalized. The initial tumor may be situated at any point of the body, except on the extremities; whether operated upon or not, it becomes associated after a few months with secondary tumors in progressive number, from about twenty to several hundreds, first in the same region, then scattered on the trunk, the root of the limbs and the head with relative freedom of the hands and feet. Some of the tumors are at first hypodermic and movable under the skin, which presents a lavender hue and an orange-peel appearance, before it becomes invaded. Other tumors are dermic, pink, dark red, then purplish, the size of a pea or a hazel-nut to that of a mandarin orange, of hard or soft

Fig. 204.—Generalized sarcomatosis. Woman, aged fifty-two years; onset in the pectoral region three years before; death in the course of the fourth year.

consistence; after a certain time, they undergo ulceration through necrosis; followed by fever, diarrhea, hemorrhages, finally death due to cachexia, after a total duration of one to four years. The glands are found to be intact, but metastases are sometimes found in the lungs, liver and spleen. The blood at the period of the ulcerations presents a leukocytosis of 20,000 to 40,000, with predominance of the polynuclears. This pathological type, known as *generalized sarcomatosis* (Fig. 204) is observed especially in patients between forty-five and sixty years of age.

Angiosarcoma is a malignant tumor, frequently multiple, developing slowly in adults, more rapidly in children, consisting of fascicular sarcomatous tissue with telangiectases; opinions differ

as to its being an angioplastic sarcoma or an angioma which has become sarcomatous. These tumors have been observed especially on the scalp, in the upper part of the face and on the upper part of the trunk.

Sarcomas with myeloplaxes are extremely rare and seem to be secondary to tumors of the bones.

Parini is said to have observed a case primary in the skin.

B. **Atypical Sarcoma with Polymorphous Cells.**—I have repeatedly drawn attention to this form of sarcoma, under the name of "lymphosarcoma:" this denomination has the disadvantage of creating confusion by suggesting the identity of this form with the glandular lymphosarcoma of Kundrat-Paltauf (p. 653); since this identity seems to me in no way established, nor even probable, I have adopted the denomination given above, in my report of an illustrative case (*Annales de Dermatologie*, April, 1911, p. 226).

This form of sarcoma is characterized histologically by a very special, alveolar or areolar structure on account of which it has received from the old anatomo-pathologists the names of *alveolar sarcoma* (Billroth) and *reticular carcinoma* (Cornil and Ranvier). The more or less dense network is sometimes adenoid, composed of fine fibrils, while in other cases it consists of larger fibrous strands, provided with connective-tissue cells; or again it may be embryonic and myxomatous; or it may vary in appearance at different points of the same tumor. The constituents enclosed in its meshes are connective-tissue cells of embryonic or fetal type and very polymorphous, being small and round, or rather large and round, or fusiform and stellate, sometimes multinuclear; among them plasmocytes are sometimes met with, but very few or no lymphoid or myeloid constituents; the polynuclears are numerous in ulcerated tumors. The bloodvessels, instead of being purely lacunar as in the other sarcomata, have distinct walls, but without elastic fibers. The boundaries of the tumor are less distinctly outlined than those of the other sarcomas.

Clinically, these polymorphous alveolar sarcomas are, after the epitheliomas, the most common primary malignant tumors of the skin. They are observed in both sexes, in adolescence or adult life, preferably in the vicinity of the natural orifices.

The process begins as a small pink intradermic nodule, of firm consistence; this develops in breadth rather than in thickness, finally giving rise to a large, hard, globular or spreading tumor, which for a long time remains solitary, more or lesss rapidly undergoing erosion or ulceration. A large and hard, sometimes also ulcerated glandular enlargement is noted at an early stage, which does not occur in any other form of sarcoma. Secondary nodules finally develop in the vicinity; generalization takes place by the lymphatic route, as in the case of the epitheliomas.

This variety is thus seen to differ in many respects from the so-called typical sarcomas.

C. **Multiple Hemorrhagic Sarcomatosis.**—This designation is applied to a disease which is clearly differentiated by its clinical behavior and by the structure of its lesions; their character, however, is probably very different from that of the sarcomas. It is the *idiopathic pigmented sarcoma of* Kaposi.

It begins at the extremities, or as a solitary tumor at any point, sometimes as the sequel of a local traumatism; lesions promptly develop on the feet and hands, nearly always symmetrically.

There are either very hard edematous swellings, livid or slate-colored, in spots or in patches, often imperfectly outlined; or small tumors, miliary or pea-sized, at first intradermic, then protuberant or even pedunculated, often agminated. These tumors, in very variable number, appear on the infiltrated surfaces as well as on the healthy skin; they vary in hue from a dark rose color to purplish and black; hemorrhagic spots are likewise noted. The lesions progress from the periphery to the center; after the legs and the forearms, the thighs, the genital organs, the mouth, the back and finally the internal organs are invaded. Movements are considerably impaired, but the pain is moderate; the tumors and infiltrations never assume a marked development, they have only a slight tendency to ulcerate and are even capable of undergoing spontaneous absorption. The glands usually remain free. The general health may be good until the terminal stage is reached.

Kaposi's sarcomatosis attacks especially men from forty to sixty years, particularly laborers, but sometimes children. It lasts from two to ten years and leads to death through generalization, with fever, cachexia, hemorrhages, etc. It is not rare in Poland, Russia, Austria and Italy, but altogether exceptional in France. [In America it occurs almost exclusively among Jews, possibly because the great majority of our Russian, Austrian and Polish immigrants belong to this race.]

Histologically, the lesions consist of a newformation of dilated capillary bloodvessels, with lymphatic dilatations and collections of round and fusiform cells arranged parallel with the vessels in variable proportions. The pigment is hemosiderin, of hemorrhagic origin.

There is a tendency to question the neoplastic character of this disease and to consider it as infectious; its pathogenic agent is unknown; it is not auto-inoculable and does not seem to be contagious.

Diagnosis of the Sarcomas.—As many sarcomas are hypodermic at the onset, their differential diagnosis must be made from all the nodosities (Chapter XIV), the gummas and especially the sarcoids,

and furthermore from the benign subcutaneous or cutaneous tumors, and from adenitis or certain subacute or chronic phlegmons.

Secondary carcinoma of the skin, nevo-carcinoma, cylindroma, mycosis fungoides, the leukemic tumors can sometimes be differentiated only with considerable difficulty.

The polymorpho-cellular sarcoma may closely resemble epithelioma, certain forms of cutaneous tuberculosis and even syphilitic chancre.

The hemorrhagic sarcoma of Kaposi must be distinguished at the onset from nevo-carcinoma, from botryomycoma, from scleroderma; and in the course of its development, from the tertiary syphilides, from lupus and more particularly from leprosy, certain cases of which have very closely simulated it in my experience.

In all doubtful cases, biopsy and histological examination as well as cytological analysis of the blood will be required for the confirmation of the diagnosis; it is advisable to resort to these procedures even when the clinical features and the course seem more or less convincing by themselves.

Treatment.—Surgical removal is to be recommended only at the very beginning, before generalization has had time to occur and must be as early and extensive as possible. In its absence, or directly afterward and as complementary measures, the following treatment should be employed:

Arsenic has at all times been universally advocated against every form of sarcoma; it is given in large doses and particularly in hypodermic injections of potassium or sodium arsenite. At the present day, intravenous injections of arsenobenzols are of course entitled to a preference. It has seemed to me as to many others, that the progress of sarcoma is delayed for some time under the influence of arsenic. [On the use of arsenic in chronic diseases see my note p. 661.]

The other treatment which has been found of value, at least in a certain number of cases, is *radiotherapy;* it has not been demonstrated that *radium* is more efficient, except in certain regions not readily accessible to the *x*-ray tube. The *x*-rays are certainly capable of diminishing the size and even of causing the absorption of sarcomas, but this is far from saying that they can effect a complete cure. Of course, large doses are administered in such cases; opinions differ concerning the necessary degree of filtration and the general plan of the treatment which notwithstanding the gravity of the condition must nevertheless be cautiously conducted. It goes without saying that these two therapeutic procedures may and should be employed concurrently.

Unfortunately, the physician is often consulted too late, when generalization is under way and all treatment has become powerless; so that sarcoma still has an extremely serious prognosis.

APPENDIX.

THERAPEUTIC NOTES.

On undertaking the treatment of a dermatosis, as with any other pathological condition, it is necessary to look into the *therapeutic indications* to begin with, as soon as a positive diagnosis has been made. What is to be done? What kind of medication may prove beneficial? By what kind of intervention may one hope to cure, that is, to restore the organism, as far as possible, to a normal condition?

The choice of the method must therefore necessarily precede the choice of the remedy. Nothing could be more unreasonable than to take up a formulary and blindly order some medicinal compound, without knowing the mechanism of its action and consequently the effects which it may produce.

The reason for the selection of a given remedy is sometimes supplied by the cause of the disease, when this is known and can be so acted upon as to remove it (examples: scabies, syphilis); in other cases it is the pathogenic mechanism which can be influenced; but more frequently, at least in dermatological practice, the lesions themselves dictate the mode of intervention, according to their nature, morphology and degree. It is really of minor importance from the therapeutic point of view, if an acute eczematiform dermatitis, for instance, be due to a physical or mechanical agent, or to this or that chemical substance; what has to be checked is the inflammation, the oozing, itching, etc.; what has to be foreseen, in order to prevent it, if possible, is the superadded infection; what must be aimed at is not to inhibit but on the contrary to favor cellular regeneration.

At the end of each section of this book, I have taken pains to indicate briefly the kind of medication adapted to the cutaneous affection or to the disease under consideration.

Here I propose to enter into greater detail concerning some of the modes of treatment.

It would be a waste of time, at the present day, to compare the relative value of internal and external treatment in dermatology. Each has its own domain, determined by the cause and nature of the

pathological process on the one hand and by the general condition of the patient on the other. No general remarks of practical value in this connection are possible.

I shall not dwell upon the different measures which constitute the armamentarium of the *general treatment* of skin diseases; these belong to the domain of general medicine. It goes without saying that the general hygiene, the cutaneous hygiene, the diet (to which a paragraph will be devoted at the end of this chapter) and even the emotional hygiene—all of which play a part in the etiology of certain cutaneous affections, require great attention and must be corrected in a considerable number of cases.

In the course of this work, I have repeatedly pointed out the advantages offered by hydrotherapy, climatotherapy, heliotherapy, watering-places, the various groups of sera, by opotherapy, vaccinotherapy, ferments and yeasts, etc.

In regard to internal pharmaceutic remedies, I have had occasion to supply some information concerning those of capital importance or most frequently employed in dermato-therapy (arsenic, arsenobenzols, mercury, etc.). A lengthy discussion of this subject would take me beyond the limits assigned to this book. I restrict myself to the reminder that the medicinal agents most frequently required include the following: the laxatives, quinine, cod-liver oil, calcium salts, phosphoric acid, phosphates and glycerophosphates, iodides and iodine-tannin preparations, chaulmoogra oil, sulphur and its compounds, iron, valerian, etc.

The *local treatment* likewise comprises various procedures or modes of action; their indications have been mentioned, but they cannot here be described in detail.

They can be classified under four headings, as follows:

1. Surgical extirpation, applicable especially to a large number of tumors, certain cases of lupus, etc.

2. Dermatological operations, curettage, scarifications, epilation, and cauterization; this technic and the results which can be obtained are admirably described in the writings of my colleague and friend Dr. Brocq, notably in his *Traité de Dermatologie pratique* (O. Doin, Paris, 1908, Vol. I), to which the reader is referred.

3. The different forms of physico-therapy (electrolysis, static electricity, Franklinization, high frequency, radiotherapy with *x*-rays, radium and radio-active substances, phototherapy, light-baths, Bier's method, treatment with hot-air or carbonic acid snow, massage, etc.). These methods are described in special text-books and it would be futile to offer a necessarily very incomplete and inadequate summary.

4. External medicinal treatment.

This chapter is devoted almost exclusively to the **external medicinal treatment.**

No complete dermatological formulary should be looked for, as I have on the contrary endeavored to incorporate in these notes only the customary substances and prescriptions or those with which I am personally acquainted. It has long been may conviction that good dermatologic treatment is practicable with the help of a very limited number of procedures and remedies. Inexperienced practitioners even more than experts in the field should refrain from experimenting with poly-pharmacal medication, but unfortunately the contrary is often the case.

The order followed in my account is that of the *remedies* which correspond to the clinical indications. Although it seems to be logical and convenient enough, the fact must not be overlooked that an arrangement on this basis possesses more apparent than real value.

Many medicinal agents, including some of the most valuable, have an unknown or hypothetical mode of action; furthermore, the same substance may have multiple effects; moreover, its action may be entirely different, or even opposite, according to the concentration in which it is employed, according to the duration of its application and according to its pharmaceutic form or its vehicle.

The *pharmaceutic form*, or let us say the physical state of an active or inert substance which is utilized as a topical agent, has a marked influence on its effects; this very important fact has been brought out by the contributions of Unna and his school. Some explanation here may not be superfluous.

The skin under normal conditions is covered with a protective layer, the horny epidermis.

The latter, composed essentially of keratine and fat, forms on its surface a sort of supple and resistant varnish, practically impermeable to water and aqueous solutions, but more or less permeable to fatty bodies, volatile substances and gases.

Perhaps the most important function of the horny layer is to oppose the evaporation of water from the body tissues; without it, an enormous loss of water would take place over a surface as extensive as that of the entire integument and at the temperature of the body. However, undoubtedly due to the sweat pores and follicles with which it is riddled, the normal skin is constantly the seat of a fairly considerable watery evaporation known as "insensible perspiration." This contributes powerfully to the regulation of the general temperature of the body and the local temperatures of the various tegumentary regions.

It is now necessary to keep in mind the modifications which this

physiological function may undergo through pathological changes of the skin on the one hand and through topical applications on the other. When the skin is congested or inflamed or when its horny covering is absent or defective, the evaporation is considerably increased. Among the topical agents, those which tend to increase evaporation are necessarily cooling and relieve congestion; whereas those which inhibit evaporation will have a heating and congestive effect.

The mode of action of the customary agents of topical treatment will be briefly examined in the following, in the light of the above data. First, however, it seems advisable to point out a mistake very frequently committed in practice, but which it is important to avoid; it consists in the application of alterative dressings, ointments or medicated plasters over crusts, scales, or hyperkeratotic layers. Even if the topical agent be judiciously selected, it must be remembered that the remedy is separated from the surface on which it is to act by a stratum or layer of dead and isolating substances. No one would dream of interposing a sheet of paper or a bit of shirt between an application and the diseased skin! *It is therefore always necessary at the start to cleanse, denude or freshen, every tegumentary lesion which it is proposed to treat.*

Water and Watery Solutions.— *Baths, douches, sprays, washes* and, better still, *poultices* and *moist occlusive dressings* whose action is more prolonged, are the most efficient and most commonly utilized cleansing agents; their general effect is moreover to relieve congestion. The macerated epidermis becomes more permeable.

Alcoholic, Ethereal Solutions, etc.—Alcohol, ether, acetone, chloroform, benzene, carbon disulphide and carbon tetrachloride, all more or less dissolve fatty substances, notably the epidermic fats; they accordingly have a cleansing and drying effect. I habitually employ a few drops of benzin to cleanse the surfaces for examination.

These bodies likewise dissolve certain medicinal substances insoluble in water; by acting as vehicles they favor their action, permitting them to penetrate down to the vicinity of the active layers of the epidermis. But the irritative effect of solutions of this kind restricts their application to a few special cases.

Powders.—By virtue of their mere physical properties, powders increase the surface of evaporation and thereby exert a cooling, drying action and relieve congestion. These effects increase with their power of absorption.

The first rank from this point of view belongs to a fossiliferous earth, known in France as "ceyssatite;" this is a natural product, an earth composed of the silicious shells of foraminifera; a small quantity of this powder suffices to transform a salve into a thick

paste. The only objection to ceyssatite is that it is not readily obtainable. [It is identical with Unna's Kieselgur or Terra silicea.] Other so-called inert powders, both mineral and vegetable, have the same properties, but to a lesser degree.

The mineral powders chiefly used are the following: talc, zinc oxide, the carbonates of calcium, magnesium or bismuth, the subnitrate of bismuth, kaolin or bolus alba; vegetable powders: corn, rice or potato starch, various flours, lycopodium. It must be kept in mind that starches and flour have the disadvantage of swelling on exposure to moisture and of undergoing fermentation.

In order to render powders more adherent, they can be incorporated in weak proportion with lotions or better in larger proportion with glycerinated or mucilaginous fluids, forming *watery pastes*. [The popular calamine lotion belongs to this class of powders in suspension in a watery fluid. Calamine is (nowadays) an artificial carbonate of zinc stained a pink color; a convenient formula is: ℞—calaminæ præp., 3.0; zinci oxidi, 4.0; glycerini, 1.0; aq. rosæ, 100.0 M. Various soluble drugs may be incorporated in this mixture; *e. g.*, boric or salicylic acid, resorcinol, etc., or other insoluble powders added, *e. g.*, sulphur precip.]

Glycerine.—Glycerine, which when pure is very hygroscopic and is miscible with water in any proportion, and its derivate, glycerite of starch, are less emollient and less cooling than watery solutions; on the other hand, when employed in dressings, they are superior to wet-dressings in that they reduce the risk of auto-inoculation with pyococci. Compared to fatty bodies they are less heating, but lend less suppleness to the horny layer.

Glycerite of starch with an addition of inert powders makes a very good paste which can be mixed with various active remedies and is easily removed by washing.

Watery Pastes.—These are mixtures of equal parts of powder and dilute glycerine. They are easily prepared, convenient and clean in use; moreover, not expensive, so that they are liked by patients and deserve their growing popular favor. They are applied in a thin layer, with a flat brush, left to dry for an instant, then covered with a generous sprinkling of talc; no dressing is necessary; they may be removed by washing with water.

The simplest type of watery paste is composed of equal parts of zinc oxide, talc, glycerine and water. The talc may be replaced by calcium carbonate or starch. A number of liquid or powdery medicinal agents can be introduced into these pastes, such as ichthyol, coal-tar emulsion, lead-water, borated water, lime-water, precipitated sulphur, etc., care being taken to observe that the proportion of glycerinated fluid and powder remains the same. The addition of $\frac{1}{2}$ per cent. of gum Arabic renders these pastes more

adherent; the addition of 5 to 10 per cent. of alcohol makes them more drying.

The various watery pastes are especially adapted to the treatment of the inflammatory and pruritic non-oozing dermatitides.

Fatty Bodies.—The fatty or oily bodies, which serve for inunctions, play an important part in dermatotherapy, lending themselves well to lasting and extensive local applications. They adhere to the epidermis and slightly penetrate it, making it supple, swelling the horny cells and detaching the scales. But they prevent the evaporation of the secretions and the cutaneous perspiration in proportion to their impermeability to water, thereby acquiring a congestive and heating effect.

In this respect, enormous differences exist between the various substances designated as fatty bodies on account of their composition. In this group belong: (1) Fats properly speaking, solid, pasty, or oily, of animal or vegetable origin (fresh or benzoinated lard, beef-marrow, whale oil, cacao butter, sweet almond oil, olive oil, castor oil, cod-liver oil), which are glycerine-ethers or triglycerides. Fats admit the incorporation of only a very small proportion of water; they are capable of saponification and turn rancid in the air. (2) Hydrocarbons, such as the vaselines, petroleum, paraffins, etc.; these are not at all miscible with water; they do not change on exposure to the air. (3) Wool fats (adeps lanæ, lainine, lanoline [eucerine] which are cholesterine ethers; large amounts of water can be incorporated into them (over 300 per cent.), transforming them into *creams*. The French Codex of 1908 designates as lanoline, wool-fat mixed with water to a proportion of 25 per 100.

The consistence of the fatty bodies may be arbitrarily varied by combining those which are liquid, such as the oils, or of slightly fluid consistence, such as lard and vaseline, with more viscid fats, like the wool-fats, or solid fats, like beef-marrow, spermaceti, cacao butter, or even with wax or paraffin. The resulting mixture retains the general properties of its constituents.

There are other additions, notably those of powders or water in considerably proportion, which modify not only the consistence, but also very notably the immediate effects of the fatty bodies.

Salves.—Salves are natural or combined fatty bodies into which one or several medicinal substances has been incorporated. As these salves are always more or less impermeable to the cutaneous perspiration, they have a heating, congestive effect, as mentioned above; but at the same time they favor the real and deep action of the active substances contained in them.

Salve Sticks.—Mixed with wax, paraffin, etc., salves may be moulded into sticks, which are easy to manipulate and very convenient.

Pastes.—By mixing the fatty bodies with a considerable quantity of powders (on the average, equal parts of fats and powders), *fatty pastes* are obtained. These soften and protect the skin surface on which they are applied; they moreover possess the essential property of being porous, permeable to perspiration, consequently relieving congestion. Medicinal substances incorporated into a paste have a moderate action, intermediate between that of salves on the one hand and of creams and watery pastes on the other.

Creams.—Creams are formed by an intimate admixture of fatty bodies with a large proportion of water or of watery solutions.

The relative quantity of water contained in the different creams is very variable; 17 per cent. in cold cream; 33 per cent. in "Galien cerate;" 43 per cent. in "cucumber cream;" 50 per cent in oil- and lime-water liniment. Mixtures of wool-fats and vaseline are best adapted to the manufacture of creams very rich in water (up to 1000 to 100) and do not turn rancid.

Creams cannot serve for active treatment, but are cooling and softening.

Milks, several varieties of which are offered in trade (notably the milk of Sapolan, the collosols, etc.), are emulsions of hydrocarbons, lanoline, etc., and possess analogous although mild properties.

Soapy salves are useful as cleansing agents.

Occlusive Dressings.—*Zinc gelatine* is permeable to perspiration and has a protective action, relieving congestion and itching.

Plasters, sparadraps or epithems, are formed by some tissue or fabric covered with a layer of a plastic substance of such consistence as to adhere firmly to the skin. The older plasters, consisting of litharge, lard and various resins, which are apt to crack and frequently have an irritating effect, are now advantageously replaced by plasters prepared with a base of lanolin or vaseline, caoutchouc and a little wax. Practically all external dermato-therapeutic agents may be incorporated into these plasters, in variable proportions. It must be kept in mind that all plasters, being impermeable, prevent evaporation and cutaneous perspiration, so that they have a heating and congesting effect.

Varnishes soluble in water (caseine ointment, gelanthum and analogous preparations) have not been adopted in current dermatological practice. [A varnish of ichthyol and water, equal parts, is valuable in many acute erythematous conditions, *e. g.*, acute dermatitides, erysipelas, etc.]

Ether varnishes (collodion), acetone (filmogen) ["new-skin"] and chloroform varnishes (traumaticine) are employed only in certain special conditions.

After this brief review of the pharmaceutic forms under which the medicinal agents used in dermato-therapy are employed, these agents will now be considered as such, arranged by their therapeutic indications. I repeat the reservations previously stated concerning the value of this classification.

Where a series of simple or compound substances appear in the same paragraph, they may be regarded as analogous; but they differ necessarily among themselves by shades which I have not always found it possible to indicate.

The somewhat complex combinations have been presented in the form of prescriptions, sometimes signed with the name of their best known author. It should be clearly understood, however, that there is nothing binding about these prescriptions; the excipients have been purposely varied, to serve as examples, so that a given excipient may always be replaced by some other of analogous consistence and the same physical properties.

The majority of the prescriptions are given on a basis of about 100 parts, as an aid to memory; it is left to the physician to estimate the quantity of remedy required in a given case.

[It may be useful to remember that 25 grams of a soft ointment, of the consistence of vaseline, is ample for a single inunction over the entire cutaneous surface of an adult; twice that amount is hardly enough to cover the surface with a firm paste like Lassar's.]

1. ANTIPHLOGISTICS.

Antiphlogistic, sedative, cooling and emollient treatment is employed in the *acute dermatitides* in general, in the *active erythemas*, *eczema*, etc. Cases in which it is indicated are therefore extremely common. It comprises several procedures between which a selection must first be made according to circumstances, but which are often employed in combination or successively.

These procedures consist of the use, under different forms, of water and watery solutions, especially to be recommended when crusted surfaces have to be cleaned at the same time; of powders and watery pastes; of creams, milks, or glycerites and more rarely, of fatty pastes or salves.

Lotions (*washes*).—Preferable to simple boiled water is an infusion of camomile flowers, lime- or elderblossoms, or a decoction of elecampane roots, marshmallow, etc., or some isotonic solution may be used, such as physiological salt solution, or better the cytoplastic solution of Professor Delbet (magnesium chloride, dry, 12.10, or crystallized magnesium chloride 25.85, to one liter of water) with which I have been especially well pleased of recent years. Weak

antiseptic and astringent solutions may also be employed (§ 2). Lotions should usually be applied hot and several times daily.

Sprays.—The above-mentioned solutions may be employed as sprays, by means of a Richardson apparatus or vapor spray, repeated several times daily.

Moist Occlusive Dressings.—These are used every day in dermatology even more than in surgery.

Moist dressings consist of compresses of sterilized gauze, soaked in one of the above-mentioned infusions, decoctions or solutions, well squeezed out so as to be merely damp, then applied so as to cover widely the affected region; they are covered with an impermeable layer of rubberized cloth, oil-silk, or gutta-percha, with a margin to spare on all sides; next, with a layer of cotton; the whole is held in place by a bandage. These dressings should generally be applied cold; they must be really occlusive and be renewed once or twice in the twenty-four hours. [The Liq. aluminii acetatis diluted with 10 to 15 parts of water is especially valuable for its astringent and mildly antiseptic properties.]

Poultices.—On account of their mucilaginous consistence, these are often better tolerated than moist dressings. They are prepared as follows: Mix with two spoonfuls of cold water, a spoonful of mashed potato or starch; slowly pour this dilution into a saucepan containing eight spoonfuls of boiling water for the mashed potato, not more than six spoonfuls for the starch, stirring to a jelly; then spread on muslin, folding the borders over the mixture. Linseed meal, the oil of which turns rapidly rancid, is not to be recommended. Ready-made starch and other poultices are on the market which only need to be softened in a little luke-warm water.

Poultices are applied cold or barely luke-warm and renewed two or three times daily; it is not usually necessary to cover them with an impermeable material. [Salicylic acid, one per cent., or boric acid, 5 per cent., may advantageously be dissolved in boiling water in preparing these poultices.]

Baths.—Aside from local warm baths, which are indicated for example in *lymphangitis*, general baths of pure water, baths of starch water, lime blossom decoctions, gelatins, etc., are oftener harmful than useful in *erythemas*, *urticarias*, *acute eczemas* and especially in the presence of a complicating *pyodermatitis*.

Alcohol Dressings.—This therapeutic procedure, inherited from Ambroise Paré, is not sufficiently appreciated. It is highly useful

and may serve at the onset to abort *furuncles* and *carbuncles, lymphangitic abscesses, bubos*, etc. The compresses should be soaked in 95° or 90° alcohol, no weaker; covered with impermeable material and renewed every twelve or twenty-four hours, according as the occlusion has been more or less perfect.

Powders.—It has been previously stated that absorbent or inert powders are far from being useless; they have on the contrary a cooling, drying action and [by increasing the surface for evaporation] evidently relieve congestion.

Various powders are often combined and coloring substances may be added to the mixture if desired by the patient, or they may be rendered slightly antiseptic or antipruritic.

Two examples follow, meeting each of these two indications:

PULVIS CUTICOLOR (UNNA).

Rice starch	40.0 gr.
Zinc oxide	25.0
Magnesium carbonate	20.0
Bolus alba	12.5
Bolus armenian	2.5

COMPOUND POWDER.

Talc. venet.	40.0 gr.
Calcii carbonat.	
Magnesii carbonat.	
Zinci oxidi	āā 20.0
Ichthyol or gomenol	1 to 2.0

Ointments.—The only ones usually to be recommended in cases of acute dermatitis are watery pastes, creams, and sometimes glycerites, etc.

Watery pastes may be simple or compound:

SIMPLE WATERY PASTES.

Zinc oxide, talc., glycerin, water	equal parts

or

Zinc oxide, calcium carbonate, glycerin, lime-water	equal parts

COMPOUND WATERY PASTES.

Zinc oxide,	
Starch	āā 25.0 gr.
Glycerin,	
Water	āā 20.0
Ichthyol	10.0
Gum Arabic, powd.	0.5

These pastes are applied with a flat brush, left to dry for half a minute, then covered freely with talc.

Among the *creams*, namely mixtures of fatty bodies and a large

proportion of water, may be quoted: cold cream, Galien cerate, cucumber cream, lime- and oil-liniment (almond oil and lime-water in equal parts). I prefer the following creams, because they do not turn rancid:

VASO-LANOLINE.

Petrolati	10.0 gr.
Lanolini anhydr.	5.0 gr.
Aq. rosæ,	
Aq. lauro-cerasi,	
Aq. aurant. flor.	āā 5.0

Triturate well, adding the waters drop by drop. If lime-water be substituted for one of the scented waters the emulsion is facilitated and keeps better.

CREAM OF STEARATE OF SODA.

Liq. sodæ	5.0
Ac. stearic	25.0
Glycerin	75.0
Aq. rosæ	120.0

Heat and mix; solidify by cooling, then heat again, stirring vigorously.

QUININE VASOLANOLINE (PREVENTIVE OF SOLAR ERYTHEMA).

Petrolati	10.0
Lanolin anhydr.	5.0
Aqueous solution of neutral bromhydrate of quinine 1 to 15	15.0

Apply before exposure to the sunlight and powder with

Talcum	15.0
Basic quinin sulphate	1.0

Certain *oils* (pyroleol of Edet, phlyctol of Robert and Carrière, which are compound purified and sterilized oils of melilotus), applied in dressings, have a soothing and truly analgesic action, valuable in *burns, phagedena* and *gangrenous ulcers.*

Starch glycerite, Sapolan cream, soapy salves and "diadermine" which is a glycerite of stearates, can sometimes substitute the creams.

Milks, Sapolan or others, which are emulsions of lanoline and hydrocarbons, are cooling and much liked by patients.

In the acute dermatitides, pastes and especially salves should be avoided; they are often very badly tolerated; fresh lard, however, is frequently very soothing.

When the inflammation is plainly subacute, simple pastes or pastes with a slight admixture of ichthyol may prove serviceable.

LASSAR'S PASTE.

Zinc oxid,	
Starch,	
Vaselin,	
Lanolin	āā equal parts

SOFT PASTE OF UNNA.

Zinc oxide,	
Prepared chalk,	
Linseed oil,	
Lime-water	āā equal parts

GLYCEROL ICHTHYOL PASTE.

Neutral glycerite of starch	70.0
Kaolin,	
Magnesium carbonate	āā 15.0
Ichthyol	3 to 5.0

ZINC, OIL AND ICHTHYOL.

Olive oil washed with alcohol	40.0
Zinc oxide	60.0
Ichthyol	1 to 3.0

2. WEAK ANTISEPTICS AND ASTRINGENTS.

It has come to be understood, especially of recent years, that antiseptics applied for the purpose of killing the microbes or inhibiting their growth, are at the same time injurious to the tissues, the more so in proportion to their activity. It is therefore not practicable to disinfect a wound or an infected cutaneous surface all at once by means of a strong antiseptic. In order to attain this end, or at least to favor the organism in its fight against the infecting agents and thereby to assist the cure, only so-called cytophylactic substances should be employed, namely substances which spare the cells; or mild antiseptics as slightly injurious to the body tissues as possible.

Lotions and Sprays.—In the majority of the cases, it is advisable in my opinion not to make an excessive use of lotions. When there is reason, however, for cleansing a wound, an ulcer, or an infected dermatosis, one of the following watery solutions may be employed: the classical borated water (boric acid 4 : 100), boricine, or biborate of soda obtained by the action of boric acid on borax, in equal parts and hot (4 to 10 : 100), dilute peroxide water, weak carbolized water (phenol 1 : 100), resorcinated water (resorcinol 1 : 200), phenosalyl or a solution of phenol salicylate (1 : 200), decoctions of cinchona, oak bark or other astringent, tannic acid decoctions, and finally a solution of lead subacetate [Liq. plumbi subacetat. dil.].

Matchless, in my opinion, is *Alibour water* [copper and zinc sulphate in camphorated water] which renders me every day the most valuable service and Labarraque's fluid, or the officinal solution of soda hypochlorite, which should be diluted with water to 4 to 10 times its volume; or better still, Dakin's solution. Burrow's solution [Liq. alumin. acetat.] is extensively used abroad.

ALIBOUR WATER (PYODERMATITIDES).

Cupric sulphate	2.0
Zinc sulphate	7.0
Camphor water saturated	300.0

Filter; for use dilute a tablespoonful or two to a glass of water.

BURROW'S SOLUTION (OOZING DERMATITIDES).

Alum	1.0
Plumbic acetate	5.0
Water	100.0

Not to be filtered; shake before use and dilute with 5 to 20 volumes of water.

[Burrow's solution is best prescribed as liq. alumini acetatis; diluted before use with 10 to 20 volumes of water.]

Moist Antiseptic Dressings.—All the above enumerated solutions may be employed for moist occlusive dressings, of course in their weakest solutions. The only exception is Alibour water, which is inefficient in dressings, in my experience. The question of the aseptic, antiseptic and other dressings will be taken up later on.

Baths.—Local baths, with mild antiseptics, may be useful in the *acute dermatitides* and *lymphangitides* of the limbs and of the penis. For general baths, I make use only of zinc sulphate (20 to 40 grams for a full bath) in the generalized *pyodermatitides.*

Solutions for Painting, Touching, etc.—Although they must not be abused, it may be useful to know the preparations which may be employed in ulcers, atonic wounds, folliculitis, herpes, etc. Tincture of iodine is really used to excess at the present time, but is nevertheless valuable for painting, as well as iodo-acetone (2 to 5 : 20) or iodo-chloroform (1 to 15), for aborting *boils* and in the *epidermomycoses.* The Lugol-Gram solution (iodine, 1 gr. KI, 2 gr., water 300 gr.) serves for *tuberculous* or *mycotic ulcers;* iodoform ether (1 : 20), guaïacol oil (equal parts), gomenol oil (5 to 20 : 100), in *tuberculous* or *sluggish ulcers;* camphorated iodoform oil (3 : 10 : 100) makes a good dressing for *soft chancres* and *bubos* (Dinet). Camphorated alcohol (1 : 100), borated alcohol (1 : 16), alcohol with resorcin (1 : 50) or alcohol with thymol (1 : 100) and borated glycerine (1 : 100) or carbolized glycerine (1 : 50) are useful for touching or brief application during a few minutes in *herpes* and *folliculitides* such as furuncles of the auditory meatus, etc.

Gargles (*stomatitis and buccal ulcerations*).—Dilute Labarraque's solution (50 : 1000), hydrogen peroxide water, "Neol" (1 : 10), a few drops of tincture of ratanhia or myrrh in a glass of water; decoction of snake-weed (bistorta) (20 : 1000); astringent gargles with alum, sodium borate, potassium chlorate; chloral hydrate (1 to 1.50 : 100), etc., with glycerine or honey.

A soapy tooth-paste should also be recommended, containing sodium borate or extract of ratanhia, etc.

GARGLE.

Sodium chlorate, Sodium salicylate, Sodium bicarbonate āā	2.0
Honey	75.0
Water	225.0

TOOTH-PASTE.

Soap		10.0
Carbonate of lime,		
Tricalcic phosphate	āā	20.0
Carmin		0.1
Oil of peppermint		3.0

Collutories.—Iodized glycerine (iodine and carbolic acid āā 0.25, KI, 1.0; glycerine 50) in *buccal ulcerations;* borated glycerine (4 : 30); or borated honey (5 : 20), for *thrush;* honey with alum (5 : 30).

Powders.—Iodoform, pure or camphorated (5 : 100) and its substitutes: iodol (iodopyrol), diiodoform (C_2I_4), aristol (diiodothymol or *thymoliodide* of Poulenc; europhen (*butocresiol*), airol (oxyiodogallate of bismuth or *alphaform*); dermatol or officinal bismuth subgallate; xeroform (tribromophenate of bismuth or *sigmaform*); chemically pure methylene blue; tannoform (*formotan*); subcarbonate of iron; cinchona, etc.

To these more or less antiseptic powders which can be prepared in mixtures, as will be shown by an example, Vincent's powder has been added since 1917, which is very efficient through the nascent chlorine given off from it and but slightly irritant; it acts as a powerful preventive of infection on recent jagged wounds; moreover it yields good results in infected wounds and ulcers of variable character and even, in my experience, in soft chancre and chancroidal bubo.

COMPOUND POWDER OF J. LUCAS CHAMPIONNIÈRE.

Iodoform,		
Benzoin powder,		
Cinchona powder	āā	100.0
Magnesium carbonate,		
Ol. eucalyptus		12.5

VINCENT'S POWDER.

Hypochlorite of lime		10.0
Boric acid, dry		90.0

Powder separately, mix well and preserve in a dry dark-glass bottle.

Ointments.—Creams, glycerites, pastes or salves may be employed, in different cases, salves being the most active; one or several of the following remedies are incorporated: Yellow oxide of Hg. (5 to 10 : 100); white precipitate (1 to 10 : 100); camphor (1 : 100); boric acid (5 to 10 : 100); salicylic acid (1 to 2 : 100); resorcinol (1 to 2 : 100); naphthol (0.5 to 1 : 100); or various essential oils: gomenol oil (5 to 50 : 100).

YELLOW SALVE (PYODERMATITIDES).

Yellow oxide of mercury,		
Oxide of zinc	āā	10.0
Resorcinol,		
Salicylic acid	āā	1.0
Vaselin		78.0

ICHTHYOLATED YELLOW PASTE (IMPETIGENIZED ECZEMA).

Ichthyol,		
Yellow oxide of mercury	āā	5.0
Resorcinol,		
Salicylic acid	āā	1.0
Oxide of zinc,		
Talcum	āā	20.0
Vaselin		48.0

Ichthyolated Lassar's paste (5 per cent.) with an addition of resorcinol (1 : 100) or salicylic acid (1 : 100) is extremely useful and in daily use; the nature of the fats or powders which enter into the composition of the excipient may be arbitrarily changed, incorporating at will reducing agents, keratoplastic or antipruritic agents, etc.

Starch glycerite with boric acid (100 : 10) is the topical application of choice in the treatment of *boils, carbuncles* and the *pyodermatitides* in general (Gallois).

Cold cream is a favorite vehicle for white precipitate (1 to 10: 100). with or without addition of tannin (1 to 5 : 100).

Borated vaseline is in common use as a weak antiseptic salve. It is also employed for the nasal fossæ.

GOMENOL–RESORCINOL SALVE (RHINITIS, FOLLICULITIS OF THE NOSTRILS).

Boric acid	10.0
Resorcinol	2.0
Gomenol	1.0
Vaselin	50.0

YELLOW EYE-SALVE (BLEPHARITIS, IMPETIGINOUS CONJUNCTIVITIS).

Yellow oxide of mercury	0.15
Guaiacol	0.05
Lanolin	6.0
Vaselin oil	10.0

Varnishes.—Antiseptic varnishes find their indications in certain dermatitides with an intact epidermis (*lymphangitis, erysipelas*); in these cases, thigenol, pure ichthyol or glycerinated liquid thiol (fluid thiol, 85; glycerine, 15) are serviceable; on the other hand as occlusives (*fissures, rhagades, ulcerations of mucous membranes*), use can be made of complex varnishes composed of various resins and balsams dissolved in alcohol or ether, which are generally found ready-made in the drug stores; such as Baume de Commandeur (Codex); steresol (Berlioz, Grenoble); adhesol (Leclerc). Varnishes having an aseptic action through their physical properties will be discussed later on.

3. STRONG ANTISEPTICS.

The statements made at the beginning of the preceding paragraph suggest that the indications for strong antiseptic agents are rare and limited.

The solutions and mixtures to be indicated in the following must under no circumstances be employed for dressings, as they would be too irritating and even caustic. They have to be reserved for washes, touching or painting.

Watery Solutions.—Bichloride of mercury or corrosive sublimate (1 to 2 : 1000); biniodide of mercury (1 : 1000); cyanide of mercury 2 to 5 : 1000; oxycyanide of mercury (5 : 1000); strong carbolized water (phenol 5 : 1000); "microcidine" or naphtholate of sodium (3 to 5 : 1000); strong hydrogen peroxide water (12 to 20 volumes) "lusoform," "aniodol," or watery, alcoholic or glycerinated solutions of formol (0.25 to 1 : 1000).

Various Solutions.—Tincture of iodine; iodized acetone (1 to 3 : 10 camphorated naphthol (2 : 1); camphorated phenol (equal parts); camphorated salol (3 : 2).

MENCIÈRE'S FLUID (LACERATED WOUNDS, ULCERS, ETC.).

Iodoform,		
Guaiacol,		
Eucalyptol,		
Bals. Peruv.	āā	1.0
Alcohol		10.0
Ether		100.0

To be used as a spray or for painting.

CALOT'S COMPOUND SOLUTION (TUBERCULOUS ULCERATIONS AND COLD ABSCESSES).

Guaiacol		1.0
Creosote		2.0
Iodoform		9.0
Camphorated naphthol		20.0
Ether,		
Olive oil (sterilized)	āā	34.0

Ointments.—The following two salves enjoy a certain popularity:

RECLUS' SALVE (SLUGGISH ULCERS) MODIFIED BY BROCQ AND H. PIED.

Phenol crystal,		
Salicylic acid	āā	1.0
Resorcinol		2.0
Camphor,		
Antipyrin	āā	5.0
Bals. Peruv.		6.0
Vaselin		80.0

Dissolve the first four substances in a little alcohol and glycerin; dissolve the antipyrin in its own weight of water; then triturate the whole in a mortar.

OINTMENT OF LUCAS-CHAMPIONNIÈRE (BURNS).

β-Naphtholate of soda	0.3
Oil of thyme,	
" geranium,	
" verbena,	
" origanum	āā gtt. xv
Vaselin	100.0

4. ANTIPRURITIC AGENTS AND LOCAL ANESTHETICS.

For the local treatment of pruritus, a series of procedures and remedies may be utilized.

Watery Solutions.—These are usually more efficient when hot, although sometimes they act better luke-warm or cold. Decoction of elecampane root (20 : 1000); bran water; infusion of chamomile blossoms (20 : 1000) or lime-tree flowers (10 to 15 : 1000); an infusion of coca leaves (10 to 20 : 1000); lime-water; cherry-laurel water; chloral hydrate (2 to 4 : 100); Labarraque's solution (20 to 50 : 100); phenol (1 to 5 : 100); saponin coal-tar (10 to 30 : 100); sapolan milk, collosol with oil of cade or cod-liver oil, etc. [Liq. picis alk. or Liq. carbonis deterg. 10 : 100.]

Baths.—These are often badly tolerated. Starch or bran baths are sometimes prescribed, with an addition of vinegar (1 liter); or baths of lime-tree flowers (1 kilo); finally, gelatine baths (250 to 500 gr. of gelatine, soften in cold water, dissolve while heating and pour into bath).

Various Solutions.—For painting or touching, the following are employed: Camphorated alcohol (1 : 10), or camphorated brandy (1 : 40), or camphorated chamomile oil (1 : 10); resorcin alcohol (2 to 5 : 100); menthol alcohol (1 to 2 : 100); or mentholated oil of vaseline (1 to 2 : 100); carbolized glycerine (phenol 5, water and glycerine, āā 50); alcoholic solutions of silver nitrate (3 to 10 : 100); lemon juice.

Especially advantageous are the alcoholic solutions of thymol (1:200), of carbolic acid (1:100), of coal-tar emulsion (5 to 10:1000), to which castor oil or glycerine (5 to 10 : 100) are added to prevent their too rapid drying; cover with powder or a watery paste.

ANTIPRURITIC SOLUTIONS.

Vinegar,	
Camphorated alcohol,	
Aq. lauro-cerasi	āā 100.0
Glycerin	50.0

Dilute with 4 to 10 parts of water.

Cocain muriate,	
Chloral hydrate,	
Resorcinol	āā 1.0
Glycerin	3.0
Alcohol	20.0
Aq. lauro-cerasi	30.0
Water	44.0

Ointments.—The application of a fatty substance on a pruritic region sometimes suffices to bring considerable relief to the patient; fresh lard, cerate or pure vaseline may serve for this purpose; the preference is often accorded to sapolan, naphtalan, pure cod-liver oil, in compound salves, or better in the form of collosol.

Needless to say, it is often very advantageous to incorporate one or several of the antipruritic substances with the salves or pastes. Use is frequently made of starch glycerite with tartaric acid (5:100), or a mentholated oil with chloroform (camphorated oil of chamomile, 100, chloroform 2 to 5, menthol 1.)

Ethyl para-amido-benzoate (anesthesine or *benzocaine* of Poulenc) is employed (10 to 30 : 100) in a lanolin cream mixed with alcohol or olive oil (10 to 30 : 100); or also in a mucilage of gum Arabic. Orthoform (*orthocaine*) should be rejected on account of the severe eruptions and intoxications which it may cause.

SALVE.

Menthol	1.0
Chloral hydrate, Camphor āā	5.0
Lanolin	35.0
Vaselin	50.0

PASTE.

Menthol	0.5
Phenol	1.0
Salicylic acid	2.0
Tumenol	5.0
Lassar's paste	90.0

COD-LIVER OIL SALVE.

Cod-liver oil	5.0 to 30.0
Olive oil	5.0 to 25.0
White wax	5.0
Paraffin	8.0
Aq. rosæ, Aq. lauro-cerasi āā	10.0

Occlusive Dressings.—Undoubtedly the mere fact of protecting the diseased surfaces from the air represents a valuable measure for the control of pruritus. Salves and pastes evidently owe to this a considerable share of their efficiency. Poultices with starch, moist dressings and properly applied cotton wraps likewise secure good occlusion, provided the affected surfaces are not too extensive. "Zinc gelatin," which is applicable to large surfaces, if desired, is still more practical; here follows the fundamental formula and a modified formula so as to render the gelatin harder in the warm season of the year:

Gelatin	15 or 30 gr.
Zinc oxide	15 or 10 gr.
Glycerin	25 or 30 gr.
Water	45 or 30 gr.

The mass is liquefied in the water-bath, applied with a flat brush, then while the coating is still viscid, the surface is dusted with absorbent cotton which adheres to it and gives it the appearance of swan-skin. A zinc-gelatin covering may be left in place for several days; it is removed by stripping or by means of warm water.

With this zinc-gelatin, ichthyol or other active substances may be incorporated; it is preferable to apply these substances by painting them on first, after which the surface is covered with the gelatin.

Varnishes and Plasters.—The entire series of tars and analogous substances may serve as a siccative coating, either in the natural state (coal-tar, vegetable-tar, ichthyol, thiol, tumenol, thigenol, etc.), or in the form of extracts, or ether and alcoholic or various other solutions.

The ordinary plasters can nearly all be utilized for the treatment of localized pruritus. For this purpose, use is made especially of zinc oxide plaster, resorcinol or phenol plaster, red oxide, or cod-liver-oil plaster, etc.

When treating pruritus and prurigo by topical medication, one must never omit adding advice concerning hygiene, hydrotherapy, the employment of electricity under various forms, hot air, hypertonic sera, etc.

Local Anesthesia.

This is very exceptionally indicated in the cutaneous neuralgias or dermatalgias (*zona, herpes,* etc.); in such cases, hot poultices, or ichthyol, guaiacol, carbolized and other solutions indicated above and painted on, are usually sufficient.

To spare the patient the pain of certain small operations such as scrapings, scarifications, rather extensive biopsies (*tuberculosis verrucosa, lupus, proliferations, tumors*), there are several methods. The quickest is freezing with a jet of ethyl chloride, which has replaced the bag of salted ice, stypage with methyl chloride, etc. Much more complete and durable is anesthesia by injection of a solution of cocaine chlorhydrate ($\frac{1}{2}$ to 2 : 100) locally or along the course of the regional nerves (fingers, penis, etc.), the patient must have eaten and be in the recumbent position; it is advisable not to exceed the dose of 0.02 at the head, 0.05 elsewhere. The addition of a few drops of adrenalin or of carbolic acid (chlorhydrate of cocaine 2, phenol cryst. 0.50 to 1.0, water 100) favors anesthesia; Schleich in his infiltration method showed that very weak concentrations are sufficient (chlorhydrate of cocaine, 0.10 to 0.20, sodium chloride 0.20, water 100). Less toxic than cocaine are allocaine, stovaine, etc.; I usually employ a solution of novocaine to which adrenalin is added just before use (novocaine 0.50 or 1.0, water

100, adrenalin $\frac{1}{1000}$, 10.0 to 25.0). It is essential after injecting analgesic agents to wait at least fifteen or twenty minutes before operating.

Bonin's mixture (chlorhydrate of cocaine, menthol, carbolic acid, āā equal parts) is very efficient when painted over mucous membranes, ulcers and excoriated surfaces.

5. CLEANSING AND KERATOLYTIC AGENTS.

This group of therapeutic agents is applicable to the *scaly* or *crusted dermatoses* and to the *keratoses*.

Baths.—To cleanse large surfaces, soapy baths may be utilized or alkaline baths (sodium carbonate 60 to 250 gr.) or sulphurous baths (hepar sulphuris 60 to 100 gr.). Vapor baths and dry air baths, at 70° or 80° (160°–175° F.) or over, act also through the sweating produced by them.

Lotions.—Any soap with a soda base may be used for soapy washes. In city practice, patients prefer a prescription of some medicated soap; ichthyol, lanolin, salicylic acid, tar, sulphur, naphthol soaps, etc., are the most commonly used.

Potash soaps, which are soft, are more keratolytic than soda soaps. Black soft soap is uninviting; but a white odorless potash soap can be made.

The alkalimetric titration of the commercial soaps varies in considerable proportions. It may prove advantageous to render them less irritating by transforming them into *super-fatted soaps*.

WHITE POTASH SOAP (VICARIO).

Cocoanut oil	200.0
Caustic potash, c.p.	70.0
Water	600.0

Saponify hot and boil down to 500. To make this soap super-fatted, add stearin and water, āā 20, to each 100.

LIQUID SUPERFATTED SOAP (FOR SURGICAL USE).

Green soap, Castile soap	āā 20.0
Olive oil	17.0
Oil of geranium or lemon	q. s.
Water	1000.0

Filter.

A decoction of quillaya (50 : 1000), quillaya extract and saponine are preferable to soaps for the cleansing of the scalp.

Gentle rubbing with a wad of absorbent cotton moistened with alcohol, ether, or better with benzine, very efficiently cleanses the skin.

An excellent measure for cleansing a cutaneous surface covered with crusts and scales consists in applying moist dressings or a poultice during several hours.

Ointments.—A simple application of vaseline, almond oil, lard, starch glycerite, a stearate such as diadermine, etc., often suffices for the softening and detachment of the scales. For the scalp, beef marrow or mixtures of fats are in favor, such as:

Cocoa butyr	30
Almond oil	70
Ol. rosæ	gtt. i

It is sometimes advantageous to make inunctions with a soapy foam which is allowed to dry upon the skin (*kerosis, acne*). Soapy ointments are very practical and can be employed on the scalp, on account of the facility of cleansing with a wad of cotton moistened in water.

SOAPY SALVE.

Powd. soap,		
Lard	āā	40.0
Almond oil		20.0
Ol. geranii		gtt. iv

Heat the fats to 150° C. (302° F.) before introducing the soap powder.

SOAP SALVE (CARLE AND BOULUD).

Lard	50.0
Potash	17.5
Water	300.0
Alcohol	10.0
Lanolin	20.0

Boil down to 120.

With these salves may be incorporated a large number of the usual medicinal substances, notably sulphur, but not bodies which are incompatible with alkalis, such as pyrogallol, chrysarobin, acids in general, mercurial salts, etc.

Soft soap in inunctions has a very powerful cleansing action (*lupus erythematodes, pityriasis versicolor*); but it is very irritating and its effects must be watched.

Salicylic acid is the keratolytic agent par excellence. In weak dosage (1 : 100), it figures in many pastes or salves as a mordant and antipruritic agent; in stronger dosage (5 : 100) in vaseline, it constitutes the most habitually used detersive agent (*psoriasis, ichthyosis, keratosis pilaris*); in benzoinated lard and castor oil (5 : 200), especially when immediately covered with an impermeable tissue, or in the form of an ointment or varnish, it displays its maximum activity (*keratoderma, circumscribed hyperkeratoses*).

Acetic acid and lactic acid also have a keratolytic action.

Resorcinol, which in weak doses acts as a keratoplastic agent, is on the contrary in strong doses (5 to 50 : 100) an excellent detersive and even exfoliating agent. The last named property is utilized in the following preparations which are destined to serve for exfoliation cures (*acne, kerosis, flat warts, pigmentations*). Resorcine may be combined with soft soap.

MIXTURE FOR FLAT WARTS.

Acetic acid (glacial),		
Lactic acid	āā	10.0
Precipitated sulphur		20.0
Glycerin		40.0

Apply cautiously; an analogous mixture may be made with ac. salicyl. or with phenol.

KERATOLYTIC SALVE FOR SEVERE ACNE OF TRUNK (BROCQ).

Resorcinol,		
Camphor	āā	10.0
Potash soap		15.0
Precipitated sulphur		30.0
Creta prep.		5.0
Vaselin		30.0

Apply for five, then ten, then fifteen minutes before a bath.

EXFOLIATING PASTE.

Resorcinol	40.0
Zinc oxide	10.0
Kaolin	5.0
Benzoated lard	25.0

Unna [modified].

EXFOLIATING MIXTURE.

Resorcinol		20.0 to 30.0
Potash soap,		
Sulphur	āā	20.0
Spts. lavandulæ		30.0 to 40.0

Plasters and Varnishes.—In the case of circumscribed keratoses and verrucosities, potash soap plasters, and especially salicylated plasters, render excellent service. Collodium is also frequently employed.

COLLODIUM FOR CORNS.

Collodii flex.	10.0
Absolute alcohol	6.0
Ether. sulph.	4.0
Salicyl. acid	2.0
Ext. canabis Indica	1.0

COLLODION FOR PAPILLARY WARTS.

Collodii flex	10.0
Absolute alcohol	6.0
Ether sulph.	4.0
Ac. salicyl.	2.0
Ac. lactic	1.0

6. BLEACHING AGENTS.

Bleaching agents are employed in the treatment of *hyperchromias, ephelides, chloasma*, etc., and of *hypertrichosis* of the face in women.

Lotions (washes).—Vinegar may be utilized, or weak solutions of hydrochloric acid (1 to 2 : 500), lemon juice, also watery or alcoholic watery solutions of sublimate (1 to 2 : 500), etc.

For hairs of the face, reliance can be placed only on applications of peroxide water repeated several times daily, or on the peroxide cream a prescription for which follows:

LOTIONS FOR CHLOASMA.

Hydrarg. bichlorid		1.0
Alcohol		q. s.
Plumbi acetat.,		
Zinci sulphat	āā	2.0
Aq. distillata		250.0
		(Hardy.)
Hydrarg. bichlorid.,		
Ammonii chlorid.	āā	0.1
Emulsion of almonds		100.0
		(Brocq.)

In this formula, tinct. benzoin 5.0 may be substituted for the ammon. chlorid.

The applications must be made in the morning and again in the evening; during the night, salves or ointments are applied.

Ointments.—Creams, pastes and salves which contain sufficiently strong proportions of hydrogen peroxide, acetic acid, mercurial salts, or sulphur and naphthol, possess a more or less strong bleaching action.

PEROXIDE CREAM

Hydrogen peroxide water	15.0
Vaselin	10.0
Lanolin	5.0
Oxide of zinc	1.0
Bichloride of mercury	0.5

F. HEBRA'S SALVE.

Calomel,		
Bismuth subnitr.	āā	6.0
Lard or vaselin		90.0

The most energetic treatment of pigmentations consists in the employment of exfoliation by means of strong resorcin pastes or mixtures [but they have only a temporary effect].

Plasters.—Mercurial, carbolized mercurial and red plaster are useful.

Whatever remedy has been employed, the treatment must be interrupted for a few days as soon as the skin becomes reddened and inflamed; the irritation should be soothed by means of a cream, or a starch glycerite with an addition of bismuth carbonate and kaolin (āā 20 : 100).

7. REDUCING AGENTS.

Under this name we include a series of extremely valuable dermatological remedies which have the common feature of being more or less avid of oxygen. This property, according to Unna, accounts for their biological action.

Weak reducing agents, or even strong reducing agents employed in weak dosage or for a short time, are keratoplastic, antiseptic, relieve congestion and itching. Strong reducing agents are exfoliating, highly irritative and give rise to severe epidermo-dermatitis.

Judging from my experience, in conformity with that of Jadassohn, the progressive order of activity of the usual reducing agents is apparently as follows: mercurial salts, resorcinol, sulphur and sulphur-containing remedies, tars (ichthyol, thiol, tumenol, coal-tar, wood-tar, oil of birch, oil of cade); the most energetic are undoubtedly pyrogallol and chrysarobin.

The therapeutic investigation of a large series of derivates of these substances, which have been extolled as less toxic and less irritative, has been carried out only outside of France and not always with the necessary thoroughness and disinterestedness. It is useful to know the names of some of these: lenigallol or *tripyrate* (tri-acetate of pyrogallol), eugallol or *mono-pyrate* (mono-acetate), saligallol (disalicylate); gallanol, eurobine or *trichrysate* (triacetate of chrysarobine), etc.

Reducing agents are employed under the most varied forms in *kerosis* and its complications, in the *dry psoriatiform* and *lichenoid eczemas*, in *psoriasis*, in the *parakeratoses* in general, in the *lichens*, etc.

In the choice of excipients and combinations, the fact must be kept in mind that the wood-tars (of pine, juniper; or cade oil, etc.), have an acid reaction, whereas mineral or coal-tar is alkaline.

Watery Lotions.—Ichthyol (sulpho-ichthyolate of ammonium) and thigenol are the only tars soluble in water. In order to render the other tars miscible with water, they must be emulsified with the assistance of saponine (saponined coal-tar) or in certain excipients such as retinol (a product of dry distillation of colophane); the opalol of Caillat and the collosol of Pépin are neutral soaps in which various tars can be emulsified; their use is clean and very convenient. The tars are also incorporated into solid soaps (ichthyol soap, tar soap, cade oil soap, etc.).

Sulphur is rarely employed dissolved; in carbon bisulphide, etc., it is dangerous; a very active commercial solution comes in cedar oil, known as denisol (7 per cent. of sulphur). The general preference is to employ sulphur either in suspension or in the colloidal state (denisoline), in lotions, either in the state of sulphides, or in soaps (sulphur soaps, sulphur-naphthol soaps).

The following two prescriptions are of every-day use:

CAMPHORATED SULPHUR LOTION (KEROSIS, ACNE, ROSACEA, ECZEMATIDES).

Precipitated sulphur	10.0
Spts. of camphor	20.0
Glycerin	5.0
Rose water,	
Water āā q. s. ad	100.0

SULPHUR LOTION (PITYRIASIS CAPITIS, SEBORRHEA).

Potassium sulphuret	2.0 to 4.0
Water	100.0

Or preferably:

Potass. sulphuret. liquid, 30 to 100 drops to a ¼ glass of hot water.

[The following is a useful modification of the well-known

LOTIO ALBA (ACNE, KEROSIS, ETC.).

Solution sat. zinci sulphatis,	
Solution sat. potassii sulphuretti āā	50.0
Spts. odorati	10.0

To this mixture, beta naphthol 0.5, or resorcinol 2.0, or sometimes glycerin 10.0, may be added.]

Soluble mercurial salts may also serve in lotions:

ACID MERCURIAL LOTION (ACNE).

Hydrarg. bichlorid	0.2
Ac. acetic	1.0
Tct. benzoin	5.0
Kaolin	5.0
Alcohol	20.0
Aquæ	70.0

MERCURIAL LOTION FOR THE SCALP.

Hydrarg. bichlorid	0.2
Ammonii chlorid (or resorcinol or chloral hydrate)	1.0
Aq. lauro-cerasi	10.0
Water	90.0

Moist Dressings.—Under this form I have employed only resorcinol (0.25 to 1 : 100) and especially ichthyol (2 to 10 : 100), with favorable results.

Baths.—There is no advantage in the employment of reducing agents in the form of baths. However, sulphur baths (hepar sulphuris 50.0 to 100.0) and the so-called Barèges baths enjoy a classical reputation.

BARÈGES' BATH.

Sodium monosulphide	60.0
Sodium chloride (dry)	60.0
Sodium carbonate	30.0

For one bath.

TAR BATH (BALZER).

Oil of cade	50 to 100.0
Fl. extr. quillaya	10.0
Yolk of one egg,	
Water	250.0

For one bath.

Alcoholic and Ethereal Lotions.—Washes of this kind are applicable in the treatment of kerotic affections of the hairless skin (*seborrhea, acne*) or more especially of analogous affections of the hairy scalp. When the scalp is very dry, castor oil (1 to 5 : 100) may be added to the alcoholic lotions. The prescriptions here given as examples may be combined in a variety of ways with stimulating mixtures.

LOTIONS FOR THE SCALP (PITYRIASIS, KEROTIC ALOPECIA).

1.	Beta-naphthol	0.10
	Hydrarg. bichlorid	0.20
	Resorcinol,	
	Ammon. chloride,	
	Chloral hydrat.	āā 0.5
	Spts. lavandulæ	100.0
2.	Hydrarg. bichlor.	0.10
	Ac. salicyl.	0.20
	Liq. carbonis deterg.	
	Spts. æther.,	
	Spts. rosmarini	āā 15.0
	Spts. vini	55.0
3.	Ol. Rusci,	
	Ol. cadini	āā 1.0
	Tct. quillayæ	20.0
	Alcohol (60 per cent.)	80.0

Ointments.—According to the degree and depth of action which it is desired to obtain, use is made of creams, pastes or salves.

The *creams* are prepared with cold cream, cerate, vasolanoline, or starch glycerite, incorporating calomel (2 to 10 : 100), turpeth mineral, yellow oxide of mercury (2 to 5 : 100) or precipitated sulphur (2 to 30 : 100).

In France, oil of cade is traditionally used in *psoriasis* in the form of glycerite:

CADE OIL GLYCERITE.	Mild.	Strong.
Ol. cadini	15.0	50.0
Saponis viridis or extr. quillaya (to emulsify)	q. s.	5.0
Glycerite of starch neutral	85.0	45.0
Ol. caryophyl	q. s.	q. s.

A glycerite can be transformed into a paste by adding to it kaolin and magnesium carbonate (āā 10 to 15 : 100); the glycerites have the advantage of permitting easy cleansing of the skin.

Pastes are more active and more convenient to use; their constituents may vary practically indefinitely. The following prescriptions are commonly employed by me (*figured* or *psoriatiform eczematides, lichenoid eczemas*, etc.):

ICHTHYOL SULPHUR PASTE.		
Sulphur precip.		3.0
Ichthyol		5.0
Resorcinol		1.0
Zinci ox., Starch	āā	8.0
Petrolati, Lanolin	āā	10.0

TAR SULPHUR PASTE.	eak.	Strong.
Sulphur. precip.	3.0	5.0
Ol. cadini	5.0	10.0
Ac. salicyl.	1.0	1.0
Resorcinol	1.0	1.0
Zinci ox., Talci āā	20.0	18.0
Petrolati	50.0	47.0

The formulas of these pastes can be well-nigh indefinitely modified by changing the nature of the powders or the fats; by replacing the ichthyol and cade oil with birch oil or other tars, or with extracts several satisfactory types of which are on the market (oleocade, oxycade); by employing other forms of sulphur, or by introducing mercurial salts. It should be remembered that except in the case of cinnabar (red mercuric sulphide), the association of sulphur and mercurials produces a black sulphide.

Salves are more particularly adapted to obstinate cases and to the treatment of certain regions.

The reducing agent is customarily introduced in a dose of 5 : 100 and the mordant (salicylic acid, resorcinol) in a dose of 1 : 100, but there are numerous exceptions.

For the scalp (*kerosis, pityriasis, seborrhea, psoriasis*), Sabouraud considers sulphur and sulphur derivatives as especially adapted to the oily forms and cade oil or its substitutes to the scaly condi-

tions. Virginia cedar oil has been shown by him to be capable of assisting or replacing cade oil, which has a very unpleasant odor, in lotions or ointments.

SALVES FOR THE SCALP AND BEARD.

1. Sulphur precip.,
   Ol. rusci . . . . . āā 5.0
   Resorcinol 1.0
   Ungt. Sapon. (Section 5) 90.0

2. Ol. cedri
   Ol. cadini deodorisati . . . . . āā 5.0
   Hydrarg. ox. flav.
   Resorcinol . . . . . āā 1.0
   Petrolati,
   Lanolin . . . . . āā 10.0
   (Sabouraud.)

FOR THE FACE (PITYRIASIS, ECZEMATIDES).

1. Calomel, vap. parat.,
   Tannin, alcoh. parat. . . . . āā 3.0
   Vaselin 100.0

2. Sulphur precipi,
   Cinnabar,
   Bals. Peruv. . . . . āā 3.0
   Vaselin 100.0

FOR THE BODY. COMPOUND SALVE (PSORIASIS).

Pyrogallol,
Ac. salicylic,
Resorcinol . . . . . āā 1.0
Sulphur precip.,
Ol. rusci,
Ol. cadini . . . . . āā 2.0
Vaselin,
Lanolin,
Adipis . . . . . āā 20 to 25.0

STRONG PYROGALLIC SALVE (OBSTINATE LICHENOID ECZEMA, PSORIATIFORM ECZEMATIDES).

Pyrogalloli 6.0
Ac. salicyl. 3.0
Picis liq.,
Ichthyol . . . . . āā 20.0
Cerati 50.0

The employment of pyrogallol salves must be very closely watched on account of the poisoning threatened by this agent. I have found that chrysarobin salves of very weak concentration (1 : 400 to 1 : 300 in lard), which have been recommended on various sides (Jadassohn, Sabouraud) are practicable when properly prepared and cautiously employed in progressive doses (**pityriasis rosea, eczematides**).

[DREUW'S OINTMENT (PSORIASIS, LICHEN SIMPLEX CHRONICUS, KERATOID ECZEMAS, ETC.).

Ac. salicylic		10.0
Ol. rusci,		
Chrysarobini	āā	20.0
Petrolati		20.0
Ol. theobromat.		5.0
Saponis viridis		25.0

(Modified.) An unscientific but powerful and effective reducing agent. To be used in courses of five days followed by a three- to five-day course of Lassar's paste.]

Certain mixtures, although containing active substances in fairly strong dosage, are often admirably tolerated, even in acute attacks of eczema. Caution in their use is nevertheless indicated; after the cutaneous susceptibility of the patient has been tested by applications of brief duration, they may be employed in permanent inunctions.

COMPOUND BALSAM (MODIFIED DURET'S BALSAM).

Resorcinol		2.0
Menthol,		
Guaiacol	āā	5.0
Ol. cadini,		
Sulphur ppti.	āā	15.0
Picis liq.		18.0
Sodii boratis		36.0
Camphor,		
Ol. ricini	āā	40.0
Glycerini		54.0
Aceton		80.0
Lanolin		100.0

LAILLIER'S SALVE (KERATODERMAS).

Sulphur ppti.,		
Ol. cadini,		
Picis liquid,		
Saponis viridis	āā	25.0

CHAULMOOGRA SALVE (PRURIGO).

Chaulmoogra oil	5.0
Sulphur	8.0
Camphor	12.0
Tar	15.0
Vaselin	62.0

Modified Baissade balsam.

Paints and Varnishes.—It is sometimes advantageous to employ reducing agents by applying them with a brush and covering with a powder, a zinc paste or a varnish. This is a practical method of employing chrysarobin; the latter is also very conveniently used in the form of *pomatum-* or *salve-sticks.*

Pyrogallol	5 to 10.0
Ether sulph.,	
Spts. vini rect.	āā 45.0

Cover with a layer of zinc-paste.

Chrysarobin	10.0
Chloroform	90.0

Cover with traumaticin:

Gutta-percha	10.0
Chloroform	90.0

CHRYSAROBIN SALVE-STICKS.

Chrysarobin	10.0
Petrolati	30.0
Ceræ	30.0
Butyr. cacao	20.0
Paraffini duri	10.0

Incorporate the chrysarobin with the vaselin, melt the other ingredients and mix. Cool in cylindrical molds.

Solutions of various tars, coal-tar as well as vegetable tars, may be prepared in alcohol-ether, or in benzol and acetone, applied with a brush and covered with a zinc paste. They are sometimes incorporated with collodion.

TINCTURE OF TAR (SACK).

Coal tar	10.0
Benzol	20.0
Acetone	70.0

Use unfiltered.

OIL OF CADE COLLODION (LICHEN PLANUS).

Collodii flex	15.0
Alcohol absolut.,	
Ether	āā 4.0
Ol. cadini	5.0
Ol. rusci	1.0

Dind (Lausanne) and Brocq have brought into vogue applications of crude coal-tar previously washed to free it from the excess of alkali.

The coal-tar is spread over the skin by means of a brush, allowed to dry, then dusted with talc powder; the application is touched up every day or every other day and the coating is allowed to act during four to eight days. Far from being as irritative as one might believe, coal-tar is as a rule readily tolerated under this form, even in cases of oozing eczema; in hospital practice, it advantageously replaces the compound balsams which are far more expensive. Unfortunately, coal-tar is not a definite product and its composition varies enormously, according to its source.

Plasters.—In the form of plasters the reducing agents exert an energetic action. The most common are mercurial, resorcinol, oil of cade and tar plasters; it is noteworthy that pyrogallol and chrysarobin plasters are not well tolerated.

8. CICATRIZING AND KERATOPLASTIC AGENTS.

A medicinal agent produces neither cicatrization nor epidermisation; these are curative effects on the part of the organism.

The physician's part is limited to: (1) protecting the cutaneous lesions against external irritants and against secondary infections; (2) putting them in the best possible condition for the fight against the microbes and for a proper repair; (3) accessorily, stimulating a sluggish repair-process.

In this respect it is greatly to the interest of the dermatologist to take into consideration the experience of operating surgeons and proceed in conformity therewith. For a number of years there was a tendency to replace antiseptic methods by the aseptic treatment of simple wounds. In the course of the great war, it was soon recognized in case of primarily or secondarily infected wounds that the at first very popular painting with iodine as well as applications of peroxide water, ether, permanganate, etc., were inadequate and even injurious.

Surgical "toilet", as timely and complete as possible, followed by progressive antisepsis, are favored at the present day.

In cutaneous pathology, conditions are different from those obtaining in surgery. I shall endeavor to arrange the problems which arise under a small number of headings.

Aseptic or Slightly Infected Lesions.—For admittedly clean surgical wounds, aseptic dressings are sufficient. Traumatic wounds, which are always infected or promptly liable to infection, burns, chemical dermatitis, etc., should be treated by one of the following measures:

Moist dressings with weak antiseptic solutions, selecting the least irritating; the so-called *cytophylactic* solutions (§1) are especially to be recommended, such as that of Prof. Delbet (Acad. des Sciences and Acad. de Méd., Sept., 1915).

Very interesting in its principle, convenient and economical, is the mechanically antiseptic *fixogen* dressing, devised by Mouchet and Loudenot (Archiv. de Méd et de Pharm. milit., Jan., 1917, p. 97). It consists of a varnish, at first known as fixol, analogous but preferable to the various mastisols, aseptofix, etc.; it is employed by painting it on the wound or the lesion and its surroundings, without preliminary disinfection; it is not irritative, agglutinates microbes, opposes their entrance and firmly fixes the layers of aseptic gauze with which it is covered after a minute's drying.

The paraffinization of wounds and burns by fusible mixtures (*ambrine*) accomplishes a non-adherent isolation, which has the fault, however, of being impermeable to secretions. I have previously stated that the dressing of burns and painful dermatitides with fixed oils to which essential oils have been added (gomenol oil, for example) or with compound oils known as pyroleol or phlyctol which are analgesic and aseptic, is preferable in my opinion.

Infected Wounds and Ulcerations.—Progressive moist antiseptic dressings, the borated sodium hypochlorite solution of Carrel-Dakin (calcium chloride 200, sodium carbonate 100, sodium bicarbonate 50, boric acid for neutralizing; the solution to be prepared extemporaneously) employed as a continuous local bath, has proved to be extremely valuable for the sterilization of war-wounds and is worthy of being likewise applied to the treatment of certain ulcers.

Dressings with the *polyvalent serum* of Leclainche and Vallée (serum of horses which have been immunized against the germs of the various suppurations), by favoring phagocytosis and regeneration of the anatomical constituents, accomplish both physiological antisepsis and cytophylaxis (Presse Médicale, April 2, 1917, p. 187).

Progressive chemical disinfection can also be aimed at by the employment of various powders. *Vincent's powder* (§2) seems to me superior to iodoform and its analogues, to ectogen, etc. Calcium chloride in the natural state, arsenobenzol as a powder, etc., are customarily employed on ulcerations of mucous membranes; methylene-blue still has its adherents.

Various antiseptic salves, the ingredients of which have been given above (see Sections 2 and 3), notably Reclus' salve, are also considered as assisting cicatrization. Colloid silver salve (10 : 100) is entitled to special mention.

Rhagades chaps, cracks.—These are treated either by mild cauterizations, or by painting them with steresol, adhesol, Commandeur's balsam, or balsam of Peru.

Sluggish Cicatrizations.—Cauterizations with silver nitrate, chromic acid, etc., will check exuberant granulations. Grafts are indicated on extensive granulating surfaces and promise a more flexible and less contracting cicatrix.

In case of sluggish ulcers, recourse may be had either to strong antiseptics, stimulants and mild caustics, or to styrax ointment and its analogues, as well as balsam of Peru. Occlusion by diachylon or medicated plasters, or by zinc-gelatin, is sometimes sufficient. Heliotherapy, light-baths, hot-air douches, high frequency currents, are sometimes of the greatest value.

It must not be neglected to place the affected limb in a suitable position and to order, according to the cases, either immobilization or on the other hand, systematic gymnastic exercises.

9. STIMULANTS AND RUBEFACIENTS.

These are employed in the *passive erythemas*, in *hyperidrosis* of the feet, in the *alopecias*, in *lupus erythematodes*, etc.

They are represented by alcohol, alcoholic preparations and tinctures, ether, chloroform, the volatile oils, camphor, mustard, iodine and the acids, which supply the basis of the majority of stimulating topical agents; the latter are almost invariably used in the form of liniments:

Liniments.—FOR FROST-BITE, HYPERIDROSIS, ETC.:

1. Tinct. iodin (or tannin)	1 to 5.0
Spts. camphor	100.0
2. Spts. terebenth.	3.0
Spts. camphor	47.0
Spts. saponat.	50.0
3. Aq. ammoniæ fort	5.0
Spts. camphor	15.0
Ol. amygdal. dulc.	80.0
4. Ac. tannici, Resorcinol, Ichthyol	āā 2.0
Glycerini	10.0

FOR STATIONARY LUPUS ERYTHEMATODES AND ALOPECIA.

Phenolis, Tinct. iodi, Chloral hydrat.	āā 5.0

Paint on the surface.

FOR ALOPECIA AREATA.

Ac. acetic glacial	1.0
Chloral. hydrat.	4.0
Etheris sulph.	30.0

Apply with gentle friction.

FOR DIFFUSE ALOPECIA.

1. Tinct. cantharidis	5.0
Tinct. jaborandi, Spts. melissæ	āā 15.0
Spts. odorati	65.0
2. Aq. ammoniæ fort.	2 to 5.0
Liq. picis alkalini, Spts. camphor	āā 15.0
Alcohol, 60 per cent.	70.0
3. Quininæ muriat	1.0
Tinct. jaborandi	20.0
Spts. ætheris	80.0
Ol. verbenæ	q. s.

The last mentioned stimulating lotions are applied on the scalp by means of a small so-called "lotion-brush." The formulas can be easily modified by incorporating into them, if indicated, one or other of the reducing agents in use for the hairy regions.

10. ANTIPARASITICS.

Vegetable Parasites.—The destruction of the *epidermophytes* is usually easy on the hairless skin (cf. pp. 519, 524, 526, 529, 531).

All the detergent remedies and strong antiseptics may be utilized, such as soft soap, naphthol, phenol, mercurial salts in solutions or in strong salves. The present preference is for iodine treatment with the tincture (1 : 20 to 50) or in a vaseline salve (1 : 100) or for chrysarobine in weak salves (1 : 300 to 1 : 3000). [For epidermophytoses of the hands and feet, Whitfield's salve (ac. salicyl 1.0, ac. benzoic 2.0, adipis benz. 12.0) is especially to be recommended.]

In case of *tinea*, chrysarobin salve-sticks may also be employed (chrysarobin 10 to 30 gr., yellow wax 20 to 40, adeps lanæ 50) or according to the formula give above.

The employment of chrysarobin on the head, in strong salves, or especially in plasters, requires great caution on account of the highly irritative action of this remedy on the conjunctivæ.

After epilation (by *x*-ray) it is advisable to rub the scalp with dilute tincture of iodine, or with carbolized glycerine (1 : 50).

Animal Parasites.—**Pediculosis.**—According to the conditions of the environment and other factors a choice should be made between the following remedies:

Powders.—Naphthaline mixed with talc (50 : 100); N. C. I. mixture of the British Army (naphthalene 96, creosote 2, iodoform2).

Liquids for Washes, Sprays or Sachets.—Benzine, xylol, petroleum, turpentine oil (15 : 1000), camphorated oil (10 : 100), spirits of camphor, carbolized water (2 : 100), chloroform water; vinegar with sublimate (1 : 500); parasiticide wash of the Hôpital Saint-Louis (sublimate 1, spirits of turpentine 130, glycerine 170, camphorated alcohol 700); cresyl solutions (for example: paracresylol or sapocresol 30, almond soap 15, water 1000, Choay).

Recently, various products derived from cresol have been recommended, as well as various volatile oils, for example:

SOLUTION FOR SPRAYS.

Anisol (methyl-phenol)	0.03 to 0.05
Denatured alcohol,	
Water	āā 50.0

Kills lice in eight to ten minutes; to be employed several days in succession.

PARASITICIDE MIXTURE (LEGROUX).

Ol. citronellæ,	
Ol. menthæ pip.,	
Ol. eucalypti glob.	āā 300.0
Naphtalin pulv.	100.0

To be used in "sachets," or in dilute alcoholic solution (5 to 100) as a wash.

Disinfection of clothing and bedding in the steam-sterilizer is of great importance; shaving of the head and body hairs, when practicable, as well as ironing the clothes with a hot iron, are to be recommended.

Salves.—These may be prepared from a large number of the above-mentioned remedies. I repeat that I have found yellow mercury salve most efficient and harmless in the treatment of **phthiriasis inguinalis** (yellow oxide of the Hg. 10, oxide of zinc, 10, salicylic acid and resorcin āā 1, vaseline 78.)

Scabies.—There is an abundance of formulas; the majority are based on sulphur.

Balsam of Peru and styrax ointment are also excellent against the itch and may be employed pure or incorporated in vaseline, or dissolved in alcohol with an addition of castor oil.

HÔPITAL SAINT LOUIS FORMULA.

Sulphur. sublimat.	20.0
Potass. carbonat.	8.0
Aq. destil.	8.0
Adipis	64.0

HELMERICH-HARDY SALVE.

Sulphuris flor.	20.0
Potass. carbonat.	10.0
Adipis	120.0

VLEMINCKX' SOLUTION.

Sublimed sulphur	250.0
Quicklime	150.0
Water	2500.0

Boil down to 1500.

WILKINSON-HEBRA SALVE.

Ol. Rusci,	
Sulphur. sublimat.	āā 10.0
Saponis viridis,	
Adipis	āā 20.0
Cretæ pulv.	5.0

BOURGUIGNON'S SALVE.

Ol. lavandulæ, cinnamonii,	
Menthæ pip., caryophyl	āā 2.0
Tragacanth	4.0
Potass. carbonat.	30.0
Flor. sulphur.	90.0
Glycerini	180.0

PERU BALSAM SALVE.

Bals. Peruv.	15.0
Styracis liq.	20.0
Cretæ præp.	20.0
Adipis (or petrolati)	45.0

With the two last named salves, disinfection of the clothing is said to be not absolutely necessary but the bedding must be disinfected.

[Sherwell's method is simple and effective: The patient, stripped and in bed, rubs himself vigorously every night with the dry powder of flowers of sulphur (sulphur sublimatum), leaving the spilled powder between the sheets and under the night-clothes. The cure requires about a week. It has the merit of simplicity and great economy, especially where an entire family has to be disinfected.]

11. CAUSTICS.

The employment of caustics for the destruction of small tumors, proliferations and nevi, is becoming more and more restricted; the preference is accorded to the galvano-cautery, superheated air and carbonic acid snow, the action of which is instantaneous and more easily graduated.

Use is still sometimes made, however, of the following: nitric, hydrochloric, lactic, trichloracetic, chromic, arsenious acids; caustic potash; zinc chloride, corrosive sublimate, acid nitrate of mercury, pure phenol, resorcin, pyrogallol, permanganate of potash, etc. Ordinary formalin has been recommended for the destruction of *proliferations*.

Arsenious acid is the remedy of choice for *epitheliomas*, in suspension in a liquid rather than in the form of a paste.

CZERNY-TRUNECEK SOLUTION.

Arsenic triox.		1.0
Aquæ,		
Alcohol (90 per cent.)	ää	50.0

Shake. For method of use see p. 688.

ARSENICAL POWDER.

Arsenic triox.	1.0
Carbonis ligni pulv.	2.0
Hydrarg. sulph. rubri	5.0

Mix with a little mucilage.

CANQUOIN'S PASTE (CARBUNCLE).

Zinci chloridi	8.0
Zinci oxidi	2.0
Amyli tritici	6.0
Aq. dest.	1.0

Make a paste.

VIENNA PASTE.

Potassii hydrox.	5.0
Calcis ustæ	6.0

Before use, make a paste by moistening with alcohol.

Silver nitrate has only a very superficial action. It is often employed in pencils or in watery or alcoholic solutions (1 : 10 to 30) on *mucous patches, erosions* and for the control of exuberant granulation of *wounds.*

Its action can be considerably strengthened by the so-called "two pencil" procedure: the surface which has been cauterized with silver nitrate is passed over with a pointed pencil of metallic zinc. This procedure is serviceable for example in cases of *ulcerated lupus.*

I have often advantageously utilized the following caustic pastes, advocated by Unna:

GREEN PASTE (LUPUS VULGARIS).

Antimonii trichlorid.		
Ac. salicylic	āā	2.0
Creosoti (Beechwood),		
Ext. cannabis Ind.	āā	4.0
Lanolin		8.0

WHITE CAUSTIC PASTE (TUBERCULOSIS VERRUCOSA).

Caustic potash,		
Quicklime,		
Green soap,		
Distilled water	āā	5.0

Apply over the lupus or verrucous patch, by means of a spatula, a layer of paste about the thickness of a knife-blade; wipe off the edges with cotton; cover with a large piece of zinc oxide plaster. In case of the white paste, it is advisable to interpolate a wad of moist cotton between the caustic and the plaster. The pain is never excessive and does not last long. At the end of a few hours the plaster is removed and moist dressings are applied. In case of the green paste, the applications are repeated twice a week on an average, until total destruction of the diseased tissue has been accomplished.

I have found these preparations preferable to cauterizations with pyrogallol, resorcin in strong salves, or potassium permanganate in concentrated solution or as a powder, which are employed by others.

12. **DIET.**

It would be altogether erroneous to assume that there is an alimentary regimen applicable to all cutaneous diseases. Nor is it a given eruption which governs the diet, but rather the general

nutritional disturbance of which it may be a direct expression or an indirect consequence or which may create a predisposition to that particular eruption.

In a patient suffering from a skin disease, the demonstration of diabetes, anemia, "lymphatic" constitution, gastro-intestinal dyspepsia or enteritis, hepatic or renal insufficiency, nervousness, or an intoxication or chronic auto-intoxication, etc., necessitates in itself the appropriate diet, such as laid down in text-books of general medicine.

Another point which must be thoroughly considered is the existence of infinitely variable, practically unlimited and often entirely unexpected individual sensibilities and idiosyncrasies of intolerance toward many articles of food and drink. It is therefore advisable to make it a rule never to ignore the statements of patients in regard to their intolerance of a given alimentary substance; on the contrary, care should be taken to guide their observations in this direction, while realizing that persons capable of trustworthy self-observation are in a great minority, as pointed out by Jadassohn.

All dermatologists have encountered patients who attribute their attacks of erythema, urticaria, pruritus or eczema, to the ingestion of strawberries, mussels, game, pork, or champagne; others hold milk, eggs, spinach, veal, etc., responsible. Although the available information on the subject of anaphylaxis is of a kind to furnish the explanation for a certain number of cases, the physician is justified in remaining unconvinced until a positive demonstration has been made.

At any rate, it would be unreasonable and exaggerated to forbid to all patients what may have harmed a few; this would result in the prescribing of an over-rigid and more or less impracticable diet.

The physician may sometimes find it necessary to recommend for a time an *exclusive regimen* such as an absolute milk diet, or a purely carbohydrate regimen, permitting only successively the addition of various kinds of foods and drinks; through this procedure, which is logical and to be recommended, one may hope to trace the idiosyncrasy of the patient. It is always advisable in such cases to be on one's guard against unintentional departures from the regimen and against fraud.

[Some knowledge of cookery is a very useful acquisition on the part of the physician. In excluding milk and eggs, for instance, it must be remembered that these articles enter into many preparations of food, various sauces, most pastries, etc.]

After these general statements, I am adding a list of the beverages and foods which are often found to be injurious to patients suffering from hyperemic or pruritic dermatoses (*erythemas, urticarias,*

eczemas, neurodermatitis, lichen planus, etc.), and which it is customary to prohibit more or less strictly.

Alcoholic beverages in general (especially unmixed wine, champagne, liqueurs, bitters, medicated wines, strong beers, cider), as well as coffee, tea and chocolate.

High meats and conserves (game, smoked meats, salted meats, sausages, meat-pies, ragouts, galantines, foie gras); certain meats, such as pork, duck, veal.

Deep-sea fish, smoked or salted fish, crabs, lobsters and shellfish.

Vegetables: Sorrel, spinach, tomatoes, stringbeans, asparagus, cabbage, cauliflower, "sour krout," cucumbers, mushrooms, truffles, raw salads.

Fruits: Strawberries, raspberries, gooseberries, melons, nuts and figs.

All cheeses, except fresh non-fermented and salted cheese. Sweetmeats and pastries.

In a general way, all condiments, vinegar, spices, mayonnaise dressings, tartare sauces, mustard, etc.

This list will be seen to comprise among others: (1) Substances possessing stimulating or irritative properties (alcohol, tea, coffee, spices); fermented, changed substances or those suspected of being so on account of their liability to change and containing toxins (high meats and venison, meat-conserves, deep-sea fish); foods of difficult digestion (salads, raw substances).

It goes without saying that restrictions or prohibitions must be rendered with tactfulness and discrimination, adapting them not only to the pathological conditions of the case but up to a certain degree to the social standing of the patient. The diet must be individualized, like all therapeutic prescriptions. Suggestions in this respect may be drawn from the few general directions which I have summarized in discussing the treatment of eczema.

[The divergence among competent dermatologists on so apparently simple a question as the value of a non-nitrogenous diet in psoriasis and some other dermatoses is certainly remarkable and suggestive. In regard to urticaria, œdema circumscriptum and similar affections which seem to be typical of an anaphylactic disturbance, how often does the most rigid inquest fail to disclose the peccant alimentary or autotoxic substance! The recently introduced "cutaneous tests" that in theory seemed so promising have proved disappointing in practice.]

In conclusion, I consider it necessary to emphasize once again the importance, which I regard as very great, of ascertaining the condition of the *teeth* (notably in *erythrosis*, in many *eczemas* and *pruriginous dermatoses*, in *alopecia*, etc. Dental caries, or the loss of a large number of teeth, an abundance of tartar, pyorrhea alveo-

laris, inflammations of the gums or gingivitis are certainly the starting-point not only of reflex disturbances but also of a real chronic intoxication. The latter is the effect either of the constant swallowing of putrid products and pus, at all hours of the day and night; or of the arrival in the stomach of imperfectly masticated and poorly insalivated food; the latter, not being in a condition to undergo in a proper way the action of the digestive juices, will permit and favor abnormal gastro-intestinal fermentations.

After having experienced it on repeated occasions, I am now convinced that putting the teeth in good condition, wearing a suitable artificial denture, with proper care of the mouth, can powerfully contribute to the inhibition of eruptive attacks, to the improvement of the general condition of many patients; while permitting the cure of skin diseases which had before resisted even correct topical applications as well as the prescription of a Draconian alimentary regimen.

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